ARCHIVES OF OTOLARYNGOLOGY

VOLUME 27 APRIL 1938 NUMBER 4

COPYRIGHT, 1938, BY THE AMERICAN MEDICAL ASSOCIATION

FACIAL PALSY OF OTITIC ORIGIN

ABRAM H. PERSKY, M.D.

PHILADELPHIA

The discussion of facial palsy in this presentation will be confined to the cases in which the otitic background is the etiologic factor. This factor may be localized in the middle ear, in the mastoid process or in both. Furthermore, it may be a complication of an acute suppurative process, a chronic suppurative process or an acute exacerbation of an old chronic suppurative mastoiditis.

One may further classify facial palsy as preoperative or postoperative.

The term "facial palsy" is used to signify palsy arising from some cause such as a suppurative or a necrotic process either in the mastoid process or in the temporal bone, a trauma during an operative procedure on the mastoid, postoperative local anesthesia or edema of the nerve from toxic causes. The condition should be carefully differentiated from true Bell's palsy, which may be due to refrigeration, toxic infection of the nerve or many other extraneous factors that may be associated with it.

Kerrison [1] stated that preoperative facial palsy occurs in association with (1) acute suppurative otitis media of the severe type in which the inflammatory process extends through the tympanic wall of the fallopian canal and the nerve either becomes involved directly or is subjected to pressure by the inflammatory products within the canal; (2) acute otitis media in which the nerve is directly exposed to the suppurative products because of a dehiscence in the wall of the facial canal; (3) chronic suppurative otitis media in which the necrosis involves the wall of the canal; (4) suppurative labyrinthitis in which the necrotic process extends through the horizontal semicircular canal to the facial canal; (5) otitic meningitis in which the facial involvement comes from the associated suppurative labyrinthitis; (6) tuberculosis of the middle ear or the mastoid process, or (7) herpes zoster auricularis (Hunt's syndrome).

Read before the Philadelphia Laryngological Society, Nov. 2, 1937.

From Dr. Coates' Otologic Service at the Graduate Hospital of the University of Pennsylvania and Oto-Rhinological Service 2 of the Mount Sinai Hospital.

1. Kerrison, P. D.: Diseases of the Ear, ed. 3, Philadelphia, J. B. Lippincott Company, 1923, p. 508.

He stated that postoperative facial palsy occurs (1) as an immediate result of division or injury of the nerve during a radical operation either reducing the wall of the posterior canal or curetting in and about the mastoid antrum; (2) as a deferred result of slight traumatism, the paralysis coming on several hours or days after the operation; (3) when the tympanic wall of the facial canal is either defective or unusually thin, when paralysis may result directly from the pressure of the gauze dressing packed too tightly into the wound cavity; (4) when mastoidectomy is performed with a local anesthetic (while the palsy is transient, it may give a great deal of concern to the surgeon, who may presuppose a more permanent injury to the nerve because of the operation; this palsy is usually noticeable before the operation is started); (5) during ossiculectomy, or (6) during operation on an infant, when if the initial cutaneous incision is carried a little too low the nerve may be injured at its exit from the stylomastoid foramen.

The course of the facial nerve exposes it to inflammation and injuries. The nerve enters the internal auditory meatus along with and above the cochlear and the vestibular division of the auditory nerve. At the fundus of the meatus it turns and traverses the petrous bone in a short narrow tunnel close to the second spiral of the cochlea to reach the hiatus Fallopii. Here the geniculate ganglion is situated, and at this point the nerve trunk bends sharply backward and outward to enter the wall of the tympanic cavity and passes on between the external semicircular canal above and the oval window below to the pyramid, where it again bends, this time passing vertically downward to emerge from the stylomastoid foramen.

In the petrous section it is exposed to injury in fracture of the base of the skull and in suppuration or necrosis of the labyrinth or the petrous bone. At the geniculate ganglion it may be involved owing to paralysis associated with herpes zoster oticus. However, it is in the wall of the tympanic cavity that the trunk is subjected to involvement most frequently—either by necrosis of the wall of the canal or by dehiscence of bone. Lastly, it may be injured, particularly in infants, at its exit from the stylomastoid foramen. A von Bezold abscess may involve the nerve at this site, particularly when the medial plate of bone is thin and the suppurative process erodes into the digastric fossa.

One can deduce a localization of the site of injury from the associated symptoms. There may be complete persistent paralysis affecting all ramifications of the nerve, in which event one may suspect a severance of the nerve or a deep-seated lesion. On the other hand, partial paralysis may occur as a result of pressure on the nerve from edema of the surrounding tissue, hemorrhagic or serous exudate, gauze or a spicule of bone in the canal or granulations over an exposed nerve. In cases of partial paralysis the various branches may be unequally affected, and one often finds that the paralysis is more pronounced in the upper branches.

The site of the injury may be further determined by disturbances of the special senses, (*a*) if there is a division or injury to the nerve in the facial canal above the exit of the nerve from the stylomastoid foramen and below the point where the branch to the stapedius muscle is given off, or (*b*) if the injury is above the branch to the stapedius muscle. In the former event the chorda tympani nerve would be involved and disturbances of taste produced, but there would be no involvement of the auditory sensations; however, one must remember that disturbances of taste may be noticed with any suppuration which is localized to Prussak's space. In the latter event there would be added a number of auditory disturbances, such as confusion of sounds, hyperacusis and tinnitus aurium.

The incidence of facial palsy is rather low, probably about 1 per cent in cases of acute otitis media and mastoiditis. (Fremel [2] reported 10 cases of complete facial paralysis in 108 cases of acute otitis media.) An analysis of tables 1 and 2 reveals that there were 31 cases of facial palsy in Dr. Coates' and Dr. Ersner's service at the Graduate Hospital of the University of Pennsylvania and at the Mount Sinai Hospital, respectively (tables 1 and 2), among approximately 1,500 cases in which mastoidectomy was performed. Hence the incidence for this group was about 2 per cent. However, if I were to limit my analysis to cases in which palsy occurred preoperatively (20 cases), the ratio would be about 1.3 per cent. There were 10 children and 21 adults in the series.

A further analysis of the tables reveals the fact that in 20 cases palsy occurred preoperatively and in 11 it resulted from some form of operation. In the first group (in which palsy preceded operation) in 8 cases palsy followed acute otitis media after a period ranging from ten days to three weeks after the onset of the otitis media (table 3). There were 4 cases in which palsy developed one or two days after the onset of an acute exacerbation of chronic otitis media. In 6 cases the involvement was associated with a chronic suppuration occurring from one month to a year after the onset of otorrhea. These last two findings are rather significant, in that they stress the seriousness of chronic infections of the middle ear. In case 12 the involvement was associated with a fracture of the temporal bone involving the mastoid process. Case 13 is interesting because the patient noticed a gradual and insidious onset, with the condition becoming progressively worse for three years. In this case there was spasticity of the muscles rather than the usual flaccid paralysis.

Summarizing the cases in which facial palsy occurred postoperatively, I find that in 5 cases the condition resulted from simple mastoidectomy, in 5 from radical mastoidectomy and in 1 from ossiculectomy. A few

2. Fremel, F.: Monatschr. f. Ohrenh. **65**:950 (Aug.) 1931.

TABLE 1.—*Palsy Occurring Prior to Operation*

No.	Patient	Age	Sex	Onset	Complications	Operation		Result
						Type	Findings	
1	B. J.	27	F	After acute mastoiditis of 2 weeks' duration	Bezold abscess	Simple mastoidectomy	Acute mastoiditis	Improved
2	M. M.	30	F	One day after acute exacerbation of chronic otitis media	None	Radical mastoidectomy	Cholesteatoma with granulation tissue over facial ridge	Improved
3	P. S.	29	M	6 days before operation after chronic discharge for 1 year	None	Radical mastoidectomy with exposure of facial nerve	Chronic mastoiditis; cholesteatoma	Improved
4	P. B.	58	F	2 weeks after onset of acute otitis media	None	Simple mastoidectomy	Acute thrombotic mastoiditis with edema of mucous membrane of deep cells along facial ridge	Improved
5	A. W.	27	F	Few days after onset of acute otitis media	None	Simple mastoidectomy	Granulation tissue; pus; soft bone	Improved
6	A. L.	53	F	Chronic otitis media 3 days before operation	None	Radical mastoidectomy	Cholesteatoma; necrosis of dural plate	Improved
7	D. C.	53	M	1 week after acute otitis media	None	None	.....	Improved
8	M. P.	11	F	Chronic otitis media 6 weeks before operation	None	Radical mastoidectomy	Exposure of facial canal	Improved
9	M. M.	49	M	Acute otitis media for 3 weeks	None	Simple mastoidectomy	Acute exacerbation of chronic mastoiditis; free pus	Slightly improved
10	B. L.	6	F	2 days after acute exacerbation of chronic mastoiditis	None	Simple mastoidectomy	Acute suppurative mastoiditis	Slightly improved
11	A. M.	63	F	10 days after acute otitis media	None	Simple mastoidectomy	Acute suppurative mastoiditis	Slightly improved
12	V. Z.	10	M	1 week after fracture of mastoid	None	Simple mastoidectomy	Removal of fragments along wall of posterior canal; nerve exposed at knee	Unimproved
13	M. M.	26	F	Insidious for 3 years	None	Radical mastoidectomy with exposure of nerve	Chronic mastoiditis with necrosis over facial canal	Unimproved
14	G. K.	50	F	1 day after acute exacerbation of chronic otitis media	None	Radical mastoidectomy with exposure of facial nerve and splitting of sheath	Chronic mastoiditis	Unimproved
15	F. S.	28	M	1 month after exacerbation of chronic otitis media	None	Radical mastoidectomy	Chronic mastoiditis	Unimproved
16	P. H.	40	M	4 months after onset of chronic otitis media	Pulmonary tuberculosis	Radical mastoidectomy	Chronic mastoiditis	Unimproved
17	M. L.	7	F	3 weeks after acute otitis media	Meningitis	Simple mastoidectomy	Acute suppurative mastoiditis	Died
18	R. S.	7	M	1 week after acute otitis media	None	Myringotomy	.....	Very slightly improved
19	R. R.	34	M	1 week before operation in course of chronic suppurative mastoiditis	Labyrinthitis (?)	Radical mastoidectomy	Previous radical revised; facial nerve completely exposed	Very slightly improved
20	S. R.	47	F	7 days after onset of acute exacerbation of chronic otitis media	None	Radical mastoidectomy	Cholesteatoma	Improved

TABLE 2.—*Palsy Due to Operation*

No.	Patient	Age	Sex	Onset	Complications	Operation		Result
						Type	Findings	
21	H. S.	54	F	1 year after simple mastoidectomy	None	Radical mastoidectomy with attempted anastomosis	Later anastomosis with hypoglossal nerve	Unimproved
22	J. W.	28	F	7 years after simple mastoidectomy	None	Radical mastoidectomy	Sclerosis	Unimproved
23	M. K.	18	F	After radical mastoidectomy	Brain abscess	Radical mastoidectomy	Exposure of dura; tapping of brain abscess	Facial palsy slightly improved
24	I. S.	50	M	After radical mastoidectomy	Brain abscess	Radical mastoidectomy	Tapping of temporal sphenoid abscess	Died
25	M. O.	28	F	After radical mastoidectomy	None	None	.....	Unimproved
26	M. B.	10	F	After radical mastoidectomy	None	None	Cholesteatoma with necrosis of dura, lateral sinus and facial canal	Unimproved
27	R. S.	33	F	After ossiculectomy	None	None	.....	Unimproved
28	S. M.	11	F	3 days after simple mastoidectomy	None	Radical mastoidectomy	Sclerosis	Unimproved
29	R. F.	11	M	After simple mastoidectomy	Septicemia	None	.....	Very slightly improved
30	T. L.	5	F	After simple mastoidectomy when semicircular canal was opened	Traumatic labyrinthitis	None	.....	Unimproved
31	R. B.	13	M	After radical mastoidectomy	Ankylosis of jaw	None	.....	Unimproved

TABLE 3.—*Causes of Facial Palsy*

Preoperative	20	Acute otitis media	8
		Acute exacerbation of chronic otitis media	4
		Chronic otitis media	6
		Fractured temporal bone	1
		Slow insidious onset	1
Postoperative	11	Simple mastoidectomy	5
		Radical mastoidectomy	5
		Ossiculectomy	1
Total			31

complications were associated with the condition in this group of cases. There was an associated von Bezold abscess in case 1. This may account for the palsy, due to involvement of the nerve at its exit from the stylomastoid foramen. In case 17 meningitis developed; in cases 23 and 24 there were abscesses of the brain, and in case 29 there was associated generalized septicemia.

The treatment varied considerably (table 4). There were 8 cases in which simple mastoidectomy was performed, 15 of radical mastoidectomy, 1 of myringotomy and 7 in which no operative procedure was employed. In 3 of the 15 cases in which radical mastoidectomy was performed the facial canal was exposed and opened, and the sheath of the nerve was split in 1 instance. In only 1 case (no. 21) was an

TABLE 4.—*Treatment*

Simple mastoidectomy		8
Radical mastoidectomy		15
Alone	11	
With exposure of facial nerve	3	
With exposure of facial nerve and splitting of sheath	1	
Myringotomy		1
No operation performed		7
Total		31

TABLE 5.—*Results of Treatment*

Improved		16
Partial	7	
Complete	9	
Unimproved		13
Died (of complications)		2
Total		31

attempt made to anastomose the severed ends of the nerve. However, at a later date, the distal end of the nerve was anastomosed with the hypoglossal nerve.

The results of these operative procedures were variable (table 5). There were 9 cases in which marked improvement or cure was noted. These were all in the group in which the involvement had been preoperative. There was slight improvement in 7 additional cases (5 of preoperative and 2 of postoperative involvement, in 1 of the latter of which there was an abscess of the brain). In 13 cases the palsy was unimproved when the patient was discharged from the hospital, and 2 patients died of complications.

A few remarks may be made as to the coincident pathologic picture, which is of secondary importance in cases of postoperative involvement but may be rather significant in cases in which the facial palsy occurs preoperatively. As noted previously, the palsy may be associated with

either acute or chronic otitis media or with an acute exacerbation of a chronic infection. In a few cases there was marked edema of the tissues about the facial ridge; granulations were evident in a number of instances and while there were no frank dehiscences, there was necrosis of the wall of the facial canal in 1 case. Careful pathologic study can furnish an index of the extent of operative procedure necessary, i. e., as to whether one is to be satisfied with simple exenteration of the diseased cells, with exposure of the facial nerve by the removal of the plate of bone or, finally, with splitting of the sheath of the facial nerve (if edema of the nerve is suspected).

CONCLUSIONS

In retrospect, a few conclusions may be drawn from this study:

1. While facial paralysis may occur preoperatively in cases of either acute or chronic suppurative otitis media, it is of greater significance and graver portent, both as to cure and as to the possibility of intracranial complication, in the latter group, especially in the presence of cholesteatoma. Hence, while one may be tempted to temporize in cases in which the involvement is acute, the situation is urgent in cases of chronic involvement.

2. In cases in which the condition is simple and acute, thorough simple mastoidectomy should be the operation of choice. In our series the results obtained from complete radical mastoidectomy with exposure of the facial nerve and subsequent splitting of the sheath did not compare favorably with the results of simple mastoidectomy. Moreover, the latter procedure should be reserved for cases in which facial palsy has occurred postoperatively.

3. Any facial palsy following operative procedure calls for an immediate exploratory operation, with an attempt to localize the site of injury and expose the injured portion of the nerve. Treatment should then be by simple drainage in cases in which there is a serous or a hemorrhagic exudate, by removal of a spicule of bone, by splitting of the sheath, by immediate repair of the severed neurons by approximation or by autoplastic graft from either proximal or distal nerves, according to the type of lesion found.

4. Finally, the duration of the palsy may be an index of the prognosis. In cases in which the palsy is of short duration favorable results may be expected of the indicated treatment. However, when the palsy has been present for a considerable period and reactions of degeneration have set in, the prognosis is necessarily more guarded, the results depending on the success of repair or other surgical procedure on the nerve itself.

1723 Pine Street.

DEVELOPMENT OF THE OTIC CAPSULE

IV. THE FOSSULA POST FENESTRAM

T. H. BAST, PH.D.
MADISON, WIS.

In 1881 Ibsen [1] published the first description of the evagination of the periotic tissue into the otic capsule back of the stapes which Siebenmann [2] in 1890 called the "fossula post fenestram." Ibsen saw this in 23 of 60 new-born children whose ears he studied. He said that there is a funnel-shaped evagination from the vestibule back of the stapes. In his figure 6 he showed projecting into it a diverticulum of the utriculus which he called the "canalis membranaceus caecus." In a few cases he could insert a human hair into the opening from the vestibule to the middle ear. Ibsen credited Cassebohm [3] with the first description of this structure. From a careful review of Cassebohm's works, such credit does not seem to be due him. While his figure 13 showed the region posterior to the stapes, he did not picture the fossula. In his text he mentioned the entrance of a blood vessel into the capsule posterior to the stapes. Ibsen, therefore, was the first to give a specific statement regarding the fossula. The diverticulum of the utriculus which Ibsen [1] showed in his figure 31*g* to project into this evagination has not been observed since.

Siebenmann [2] wrote:

> Between the posterior ampulla and the posterior end of the oval window there is in about half of my corrosions—in the new-born much oftener than in the adult—a small pyramid-like structure which projects into the bony capsule.

He compared it with the funnel-like structure described by Ibsen.

I made the next reference to this structure [4] in 1930. Since then I have had occasion to study serial sections of nearly 100 ears of various ages, and a more accurate account of the structure can be given.

From the Department of Anatomy, the University of Wisconsin.

These studies were in part supported by the Research Council of the American Otological Society.

1. Ibsen, I.: Anatomiske undersøgelser over ørets labyrinth, edited by P. L. Panum, Copenhagen, H. Hagerup, 1881. This article was written by Ibsen in 1846.

2. Siebenmann, F.: Die Korrosions-Anatomie des Knöchernen Labyrinthes des menschlichen Ohres, Wiesbaden, J. F. Bergmann, 1890.

3. Cassebohm, I. F.: Tractus quatuor anatomici de aure humana, Halae Magdeb, 1734, pp. 19, 45 and 15.

4. Bast, T. H.: Ossification of the Otic Capsule in Human Fetuses, Contrib. Embryol. **21**:53 (June) 1930.

MATERIALS AND METHODS

This account is based on a study of serial sections of 77 ears of human fetuses ranging in age from 8 weeks to full term and 20 ears of children ranging in age from birth to 6 years. Two or three representative sections through the fossular area of each ear were photographed, and a number of ears were modeled, to show the position of the fossula in relation to the surrounding structures. The photographs of the sections of each ear were mounted on one card. These cards were a great aid in classifying the types of fossulae.

OBSERVATIONS

The fossula post fenestram is an evagination of the periotic tissue extending into the bony capsule just posterior to the oval window, at a point about one third of the way between the oval window and the nonampullated end of the lateral semicircular canal (figs. 1 and 2). It may be compared with the fissula ante fenestram [5] in character and content. It differs from the fissula ante fenestram in that its shape is more sacklike and that as a rule it opens only from the vestibule and does not extend to the middle ear, although in a few cases it does communicate with both the vestibule and the middle ear. The fossula post fenestram is not a constant structure, as may be noted from the account of Ibsen, who found it in 23 of 60 new-born children. In my collection of ears the percentage ran somewhat higher, but this no doubt was due to the fact that I included small fossulae and those which were blocked with cartilage. No doubt Ibsen, with his method of study, missed these. Table 1

TABLE 1.—*Percentage of Ears Having a Fossula*

Ears from:	Number Studied*	Number with a Fossula	Percentage with a Fossula
Fetuses	64	44	68.75
Fetuses at term	9	6	66.67
Children	20	13	65

*Four ears which were too young for the fossula to be developed are not listed here.

gives the number of ears studied and lists the percentage and the number of ears with a fossula post fenestram.

The percentages indicate that the fossula is present slightly more often in fetal than in older ears. Of course, the total number of ears was too small to warrant drawing accurate conclusions from the percentages; nevertheless, the indication is substantiated by the fact that in late fetal life a few ears have a fossula which is filled with cartilage or young bone. This suggests that in a small number of older ears the

5. Bast, T. H.: (*a*) Development of the Otitic Capsule: II. The Origin, Development and Significance of the Fissula Ante Fenestram and Its Relation to Otosclerotic Foci, Arch. Otolaryng. **18**:1 (July) 1933; (*b*) III. Fetal and Infantile Changes in the Fissular Region and Their Probable Relationship to the Formation of Otosclerotic Foci, ibid. **23**:509 (May) 1936.

fossula may be so completely ossified that it cannot be identified. This point will be referred to under the section entitled "Comment."

Origin of Fossula Post Fenestram.—The youngest ear in my collection which clearly showed a fossula post fenestram was in embryo 22, of

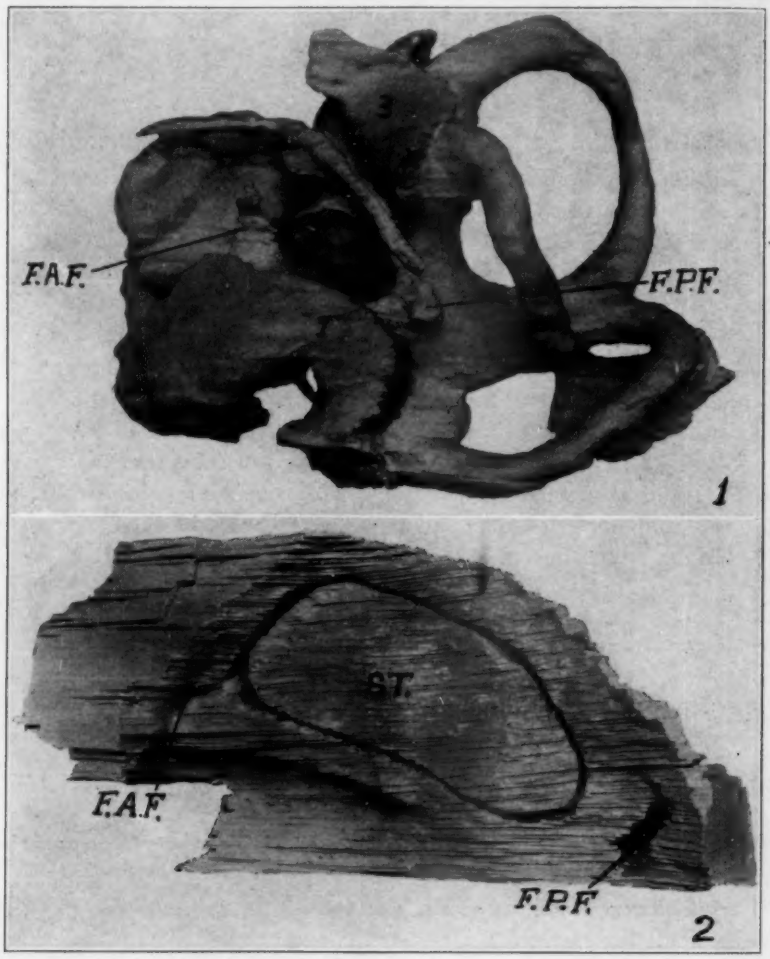
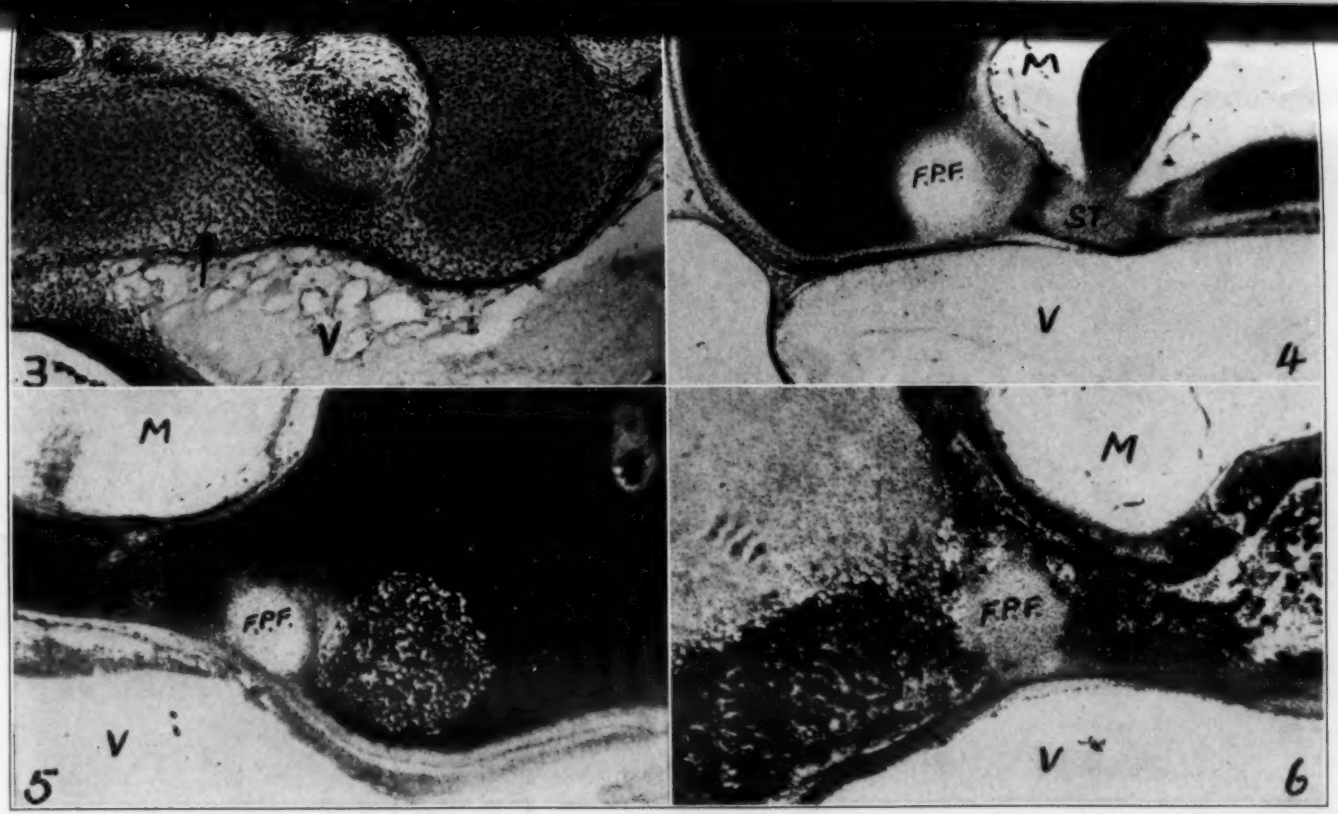


Fig. 1.—Model of the left ear of embryo 38 (147 mm. crown-rump length), aged 18 weeks, showing the fossula post fenestram (*F. P. F.*), the fissula ante fenestram (*F. A. F.*) and centers of ossification 1 and 3.

Fig. 2.—Model of the stapedial region of the otic capsule, viewed from the vestibule and showing the stapedial footplate (*ST.*), the fissula ante fenestram (*F. A. F.*) and the fossula post fenestram (*F. P. F.*).

100 mm. crown-rump length and aged about 14 weeks (fig. 4). In this ear the character and the structure of the tissue of the fossula post fenestram were much the same as those of the fissula ante fenestram,



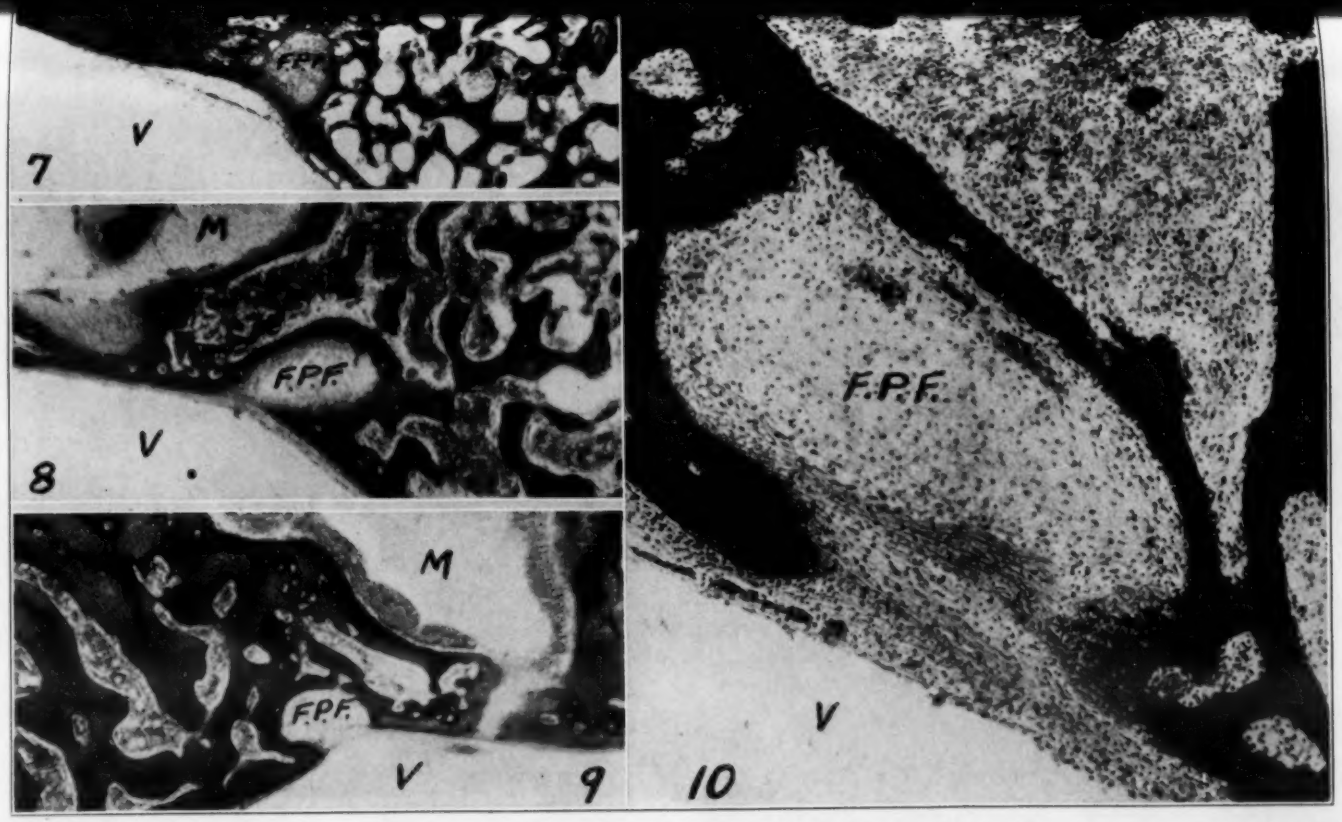
Figs. 3-6.—Photographs of sections through the fossular region of the ears of human embryos. In figure 3 (embryo 86, 50 mm. crown-rump length, age 8 weeks) the arrow points to a dedifferentiating precartilage. This is the point at which in older ears the fossula post fenestram occurs. Figure 4 (embryo 22-L, 100 mm. crown-rump length, age 15 weeks) shows a young fossula which is not yet clearly outlined from the surrounding cartilaginous capsule. Figure 5 (embryo 30-R, 146 mm. crown-rump length, age 18 weeks) shows an early process of ossification adjacent to the fossula post fenestram. In figure 6 (embryo 41-L, 160 mm. crown-rump length, age 19 + weeks) the process of ossification surrounds the fossula. *M* indicates the middle ear; *V*, the vestibule; *F. P.F.*, the fossula post fenestram, and *ST.*, the stapes.

which would indicate that both structures were approximately at the same stage of development.

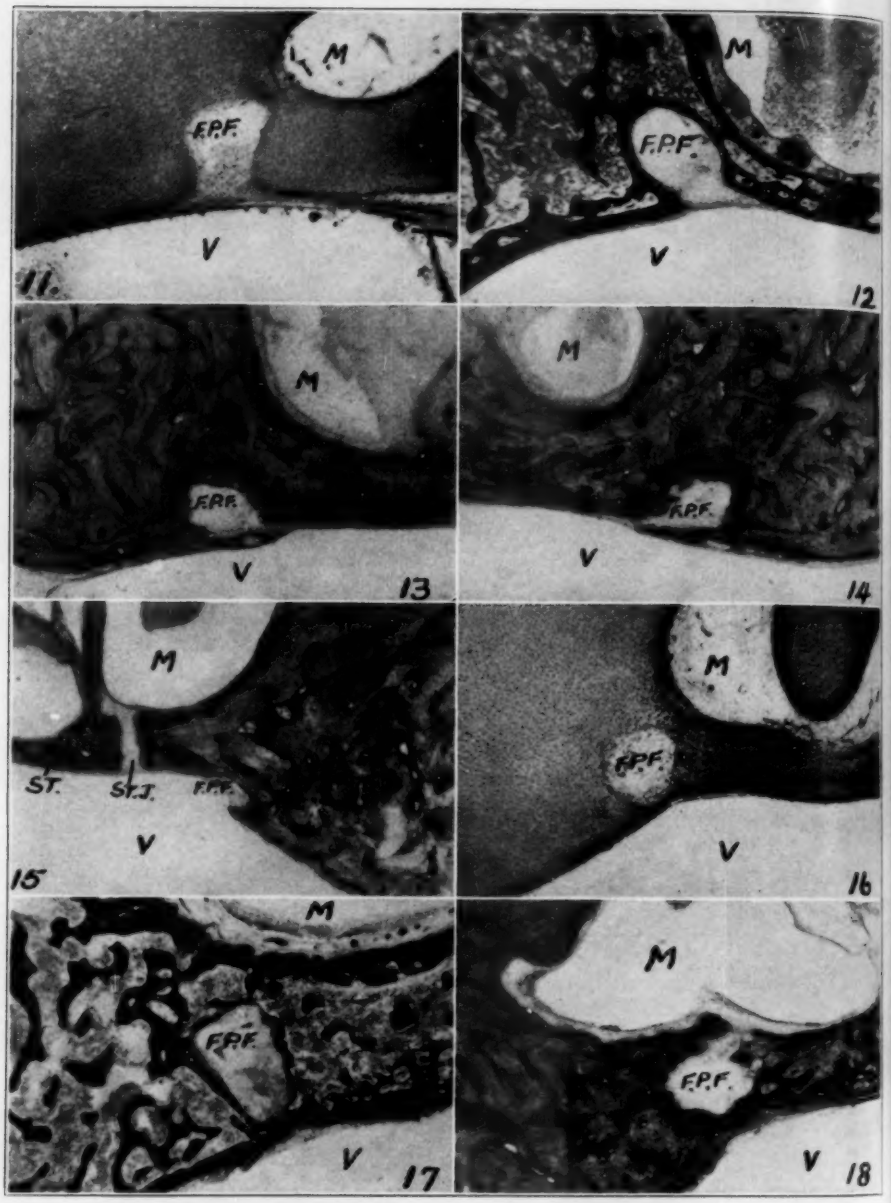
The earliest indication of the fissula ante fenestram was found in embryo 86, of 50 mm. crown-rump length and aged 8 weeks.[5a] Although in older ears the fossula post fenestram seems to be at the same stage of development as the fissula, no distinct fossula could be seen in the 8 week old embryo. However, in the region where it should be there was rarefaction of the precartilage, and an early cartilage surrounded it (fig. 3). Since this area of rarefaction was not definitely circumscribed and was not in direct communication with the vestibular tissue, it cannot be said with certainty that it was an early fossula. Then, too, there is the possibility that this ear was one of the 33 per cent in which the fossula post fenestram never develops. Because of this uncertainty and because I have no access to other ears of this early age, the details of the origin of the structure cannot be given here. However, because the subsequent course of development almost parallels that of the fissula ante fenestram, one is tempted to guess that its origin is much the same. I discussed the origin of the fissula ante fenestram in another communication.[5a]

Development of Fossula and Surrounding Capsule.—The histologic details of the development of the tissue of the fossula post fenestram are so nearly like those of the fissula ante fenestram [5a] that they need not be described here. In brief, the precartilage in this area dedifferentiates into a loose mesenchymal type of tissue (fig. 10), which later becomes slightly fibrous. This fossular tissue in most cases communicates with the vestibular tissue as it does in the fissula ante fenestram, but it differs from the latter in that it opens into the middle ear in only a small percentage of cases. When it does open into the middle ear, it receives blood vessels from the middle ear, as is the case in the fissula ante fenestram (fig. 18).

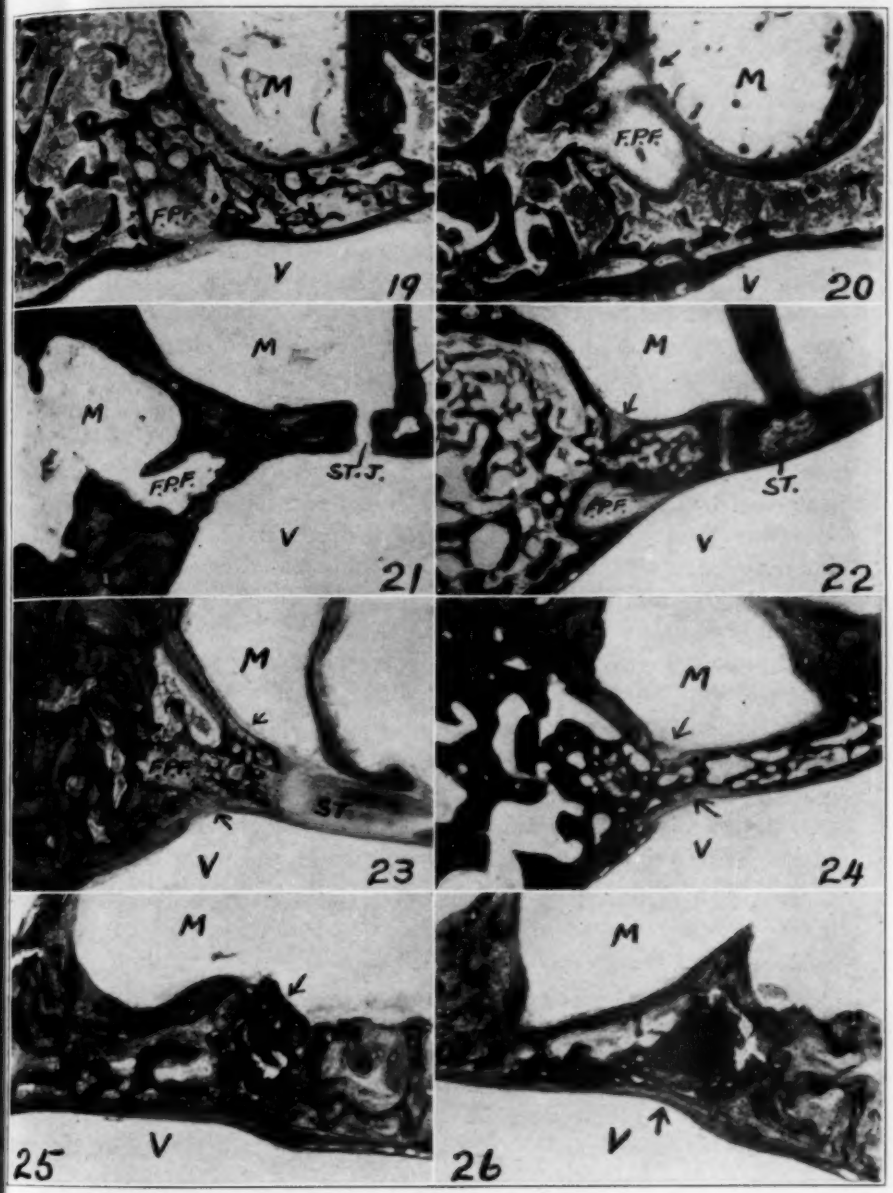
In the early stages of development the fossula is not clearly delimited from the surrounding cartilaginous capsule (fig. 4), but shortly thereafter (figs. 11 and 16) it is clearly outlined by the capsular cartilage. In fetuses of 145 to 150 mm. crown-rump length (age, 18 weeks) the cartilage in this area begins ossification. This process is pictured in figures 3 to 7. Ossification is accomplished by the extension of the center of ossification numbered 1 (fig. 1) in its growth upward to meet the center of ossification numbered 3.[4] In fetuses of 180 mm. crown-rump length (age 22 weeks) the fossula is well surrounded by intrachondrial bone (fig. 7), and in a fetus of 230 mm. crown-rump length (age 26 weeks) the fossula is definitely encapsulated by periosteal bone (fig. 8). At the age of 32 weeks the intrachondrial bone (so-called cartilage islands) of the capsule is well encased by endochondral bone (fig. 9). During this



Figs. 7-10.—Photographs of sections through the fossular region of the ears of human embryos, showing successive stages of ossification of the otic capsule around the fossula post fenestram. In figure 7 (embryo 29 B-R, 180 mm. crown-rump length, age 21 + weeks) the fossula is surrounded by intrachondrial bone and bone marrow. In figure 8 (embryo 2-R, 230 mm. crown-rump length, age 26 weeks) the fossula is encapsulated by perichondral bone. In figure 9 (embryo 110-L, 290 mm. crown-rump length, age 32 weeks) the otic capsule consists of three types of bone. The darkly stained spicules are intrachondrial bone. These are surrounded by the lighter-stained endochondral bone. Perichondral bone is forming around the fossula post fenestram, along the middle ear and along the vestibule. Figure 10 (embryo 51-L, 210 mm. crown-rump length, age 24 weeks) is a high power view of the fossula, showing the mesenchymal type of tissue which fills it. Three blood vessels may be seen in the upper part of the fossula and the vascular channels which extend between the fossula and the bone marrow, through the bony capsule which surrounds the fossula. *M* indicates the middle ear; *V*, the vestibule, and *F. P. F.*, the fossula post fenestram.



Figs. 11-18.—Photographs of sections through the fossular region of the ears of human embryos, showing different types of fossulae. Type 1, a large fossula with a wide opening to the vestibule, is shown in figures 11 (embryo 12-L, 135 mm. crown-rump length, age 17 weeks) and 12 (embryo 114-L, 230 mm. crown-rump length, age 26 weeks). Type 2, a large fossula with a small opening to the vestibule, appears in figures 13 and 14 (left and right ears of baby 93, age 14 months). Type 3, a fossula consisting of a shallow depression from the vestibule, is shown in figure 15 (baby 96-R, age 3 years). Type 4, a large fossula the vestibular opening of which has been obliterated, appears in figures 16 (embryo 5-L, 135 mm. crown-rump length, age 17 weeks) and 17 (embryo 42-L, 240 mm. crown-rump length, age 27 weeks). Type 5, a large fossula opening to the middle ear only, is illustrated by figure 18 (baby 103-L, age 1 year 10 months). *M* indicates the middle ear; *V*, the vestibule; *F. P. F.*, the fossula post fenestram; *ST.*, the stapes, and *ST. J.*, the stapedial joint.



Figs. 19-26.—Photographs of sections through human ears, showing types of fossulae. Type 6, in which the fossula extends through the otic capsule from the vestibule to the middle ear, is illustrated by figures 19 and 20 (embryo 112-L, 240 mm. crown-rump length, age 27 weeks), figure 19 showing the vestibular opening and figure 20 the opening of the middle ear, and by figure 21 (baby 120-L, age 7 months), which shows the fossula opening into the middle ear. Sections of this ear taken at a little lower level show the opening into the vestibule. Type 7 is a saclike fossula definitely opening into the vestibule but not into the middle ear, although there is a definite depression from the side of the middle ear, indicated by the arrow in figure 22 (embryo 46-L, 222 mm. crown-rump length, age 25 weeks). Type 8 is illustrated by figures 23 to 26. The arrows indicate openings from the vestibule and from the middle ear into the fossula, but the fossula proper is obliterated by either cartilage or bone. In figure 23 (embryo 123-L at term) the fossula is filled with cartilage which is beginning to ossify. Figure 24 shows the fossula of embryo 122-L (350 mm. crown-rump length, age 38 weeks) and figures 25 and 26, that of embryo 67-R (370 mm. crown-rump length, at term). *M* indicates the middle ear; *V*, the vestibule; *F. P. F.*, the fossula post fenestram; *ST.*, the stapes, and *ST. J.*, the stapedial joint.

entire process of ossification of the capsule the tissue of the fossula post fenestram retains its loose embryonic character, as shown in figure 10.

Blood Supply of Fossula Post Fenestram.—That the fossula is supplied with blood may be seen in figure 10. The three vessels in the upper part of the fossula were at first thought to be branches of the vessels of the vestibule shown in the lower part of the picture. A careful study of the serial sections revealed that these fossular vessels communicate with the vessels of the neighboring bone and bone marrow. Two such vascular channels are seen in the bone (fig. 10) above the fossular vessels. These channels extend through the bone between the bone marrow and the fossula. Thus, in spite of the definite capsule of bone which surrounds the fossula, there are channels of communication between it and the bone marrow. In some cases, as is shown in figures 19 and 20, there are wide communications between the fossula and the bone marrow.

In those cases in which the fossula opens into the middle ear, blood vessels enter it from the membranes lining the middle ear.

Types of Fossulae.—There is considerable variation in the size and the shape of the fossula post fenestram. Its communication with the vestibule or the middle ear is inconstant. On the basis of these variations the fossulae may be placed in three main groups, and these may be subdivided into eight types. These groups and types are as follows:

Group I: The fossula post fenestram communicates with the vestibule only. Seventy-three and one-third per cent of the fossulae studied came under this group. This group of fossulae may be subdivided into four types:

1. Those (23.33 per cent) which are large and have a wide communication with the vestibule (figs. 11 and 12).

2. Those (13.33 per cent) which are fairly large but have a small, slit-like communication with the vestibule (figs. 13 and 14).

3. Those (15 per cent) which consist of only a shallow outpocketing from the vestibule (fig. 15).

4. Those (21.67 per cent) which are large but for which the communication with the vestibule is almost obliterated (figs. 16 and 17).

Group II: The fossula communicates with the middle ear only. Under this group there is only one type:

5. Those (rare) which are large but for which the communication with the middle ear is small (fig. 18). Only one ear in my collection had this type of fossula.

Group III: The fossula communicates with both the vestibule and the middle ear. Twenty-five per cent of the fossulae studied belonged to this group, and they were of three types:

6. Those (15 per cent) in which the evagination from the vestibule and that from the middle ear communicate, so that the fossula extends from the vestibule through the otic capsule to the middle ear (figs. 19-21).

7. Those (5 per cent) in which the fossula is indicated as an evagination both from the middle ear and from the vestibule but in which these do not communicate (fig. 22).

TABLE 2.—*Classification of Types of Fossulae*

	Human Fetuses				Infants			Totals	
Types	Catalog No.*	Crown-Rump Length, Mm.	Approx. Age in Weeks	Sex	Catalog No.	Age	Sex	No. of Ears	Percentage
	Group I. Opening to Vestibule Only; 73.33%								
1. Wide opening and large pocket	12-L	135	17	Male					
	30-R	146	18	Female					
	38-L	147	18	Male					
	40-L	155	19	Male					
	41-L†	160	19+	Male					
	29B-R	180	21+	Female					
	21-L	183	21	Male					
	47A-L	210	24	Male					
	51-L	210	24	Male					
	62-L	215	24+	Female					
	114-L	230	26	Male					
	113-L	235	26+	Male					
	116A-L	280	31	Female					
	110-L	290	32	Male	.....	.....	.....	14	23.33
2. Narrow opening but good-sized pocket	2-R	230	26	.....	98-L	6 mo.	Male		
	80-L	230	26	Female	78-L	7½ mo.	Male		
	108-L	285	31+	Male	97-L	10 mo.	Male		
					93-R	14 mo.	Male		
					96-L	14 mo.	Male	8	13.33
3. Wide opening and shallow pocket	53-L	112	15+	Male	115-L	New-born	Female		
	54-L	115	15+	Female	96-L	3 yr.	Male		
	37-L	140	17+	Male	96-R	3 yr.	Male		
	45B-L	180	29(?)	Male					
	45A-L	260	29	Male					
	116B-L	280	31	Female	.....	.....	.....	9	15.00
4. Opening almost or completely obliterated, but large pocket	22-L	100	15	Female	95-L	New-born	Male		
	55-L	117	15+	Male					
	5-L	135	17	Male					
	36-L	135	17	Male					
	39-L	150	18	Male					
	33-L	163	20—	Female					
	42-L	240	27—	Female					
	111-L	255	28+	Female					
	107-L	270	30	Female					
	109-L	280	31	Male					
	59-L	290	32	Female					
	66-L	290	32	Male	.....	.....	.....	13	21.67
	Group II. Opening to Middle Ear Only; 1.66%								
5. Small opening and large pocket	.....	...	..	.....	103-L	1 yr. 10 mo.	Male		
						20 days	.....	1	1.67
	Group III. Opening to Vestibule and Middle Ear; 25%								
6. Openings communicate	57-L	112	15	Male	124-L	4 days (premature)			
	85B-L	198	22	Female					
	91-L	220	25	Male	83-L	10 wk.	Male		
	112-L	240	27	Female	120-L	7 mo. 13 days	Male		
	16-L	363	40	Female	118-R	5 yr.	.....	9	15.00
7. Openings do not communicate	46-L	222	25						
	85A-L	225	25+						
	60-L	305	33+	Female	.....	.....	.....	3	5.00
8. Pocket plugged with cartilage or bone	122-L	350	38	Male	67-R	New-born	Male		
					123-L	New-born	Male	3	5.00

* The letters A and B after catalog numbers indicate twins. L and R indicate left and right ear.

† The pocket of the fossula was filled with young cartilage cells.

8. Those (5 per cent) in which the pattern of the fossula is much like that in type 6 but the pocket of the fossula is plugged with either cartilage or bone (figs. 23-26).

This classification is followed in table 2.

COMMENT

The types of fossulae described are purely developmental and are anatomic, not functional, types. They are more variable and less constant than the fissulae. This suggests that the fossula is a more rudimentary structure and probably less significant from the functional point of view than the fissula.

In a previous paper I [5b] described certain changes in the fissula ante fenestram which lead to partial or complete obliteration by chondrification and later by calcification and even ossification. Some of the areas of ossification resemble otosclerotic bone. It was suggested that these changes are the early steps in the formation of otosclerotic foci. It is of interest that in a small percentage of cases such changes were noted in the fossula. The earliest example of this change is shown in figure 6, in which the section is from the ear of a fetus of 160 mm. crown-rump length. Here the fossula is clearly seen, but the tissue is young cartilage. In figure 23 the cartilage is much older, and ossification occurs at the periphery. In figures 24 to 26 the area is ossified, but the character of the bone is different from that of the surrounding bone. This, then, suggests an unstable area in the otic capsule, which may later change into otosclerotic bone. If these changes are the early changes in formation of otosclerotic bone, then the fact that these changes are less frequent in the fossular than in the fissular area would account for the fact that otosclerotic bone is usually found in the otic capsule anterior to the stapes.

SUMMARY

1. The fossula post fenestram is an evagination of the vestibular tissue into the bony capsule posterior to the stapes. Its origin and development are much like those of the fissula ante fenestram.

2. It occurs in about 67 per cent of all ears studied. It extends through the otic capsule from the vestibule to the middle ear in 25 per cent of the ears having a fossula. In about 1.67 per cent it opens to the middle ear only, and in the other 73.33 per cent it opens to the vestibule only.

3. Cartilaginous and bony changes in the fossula occur in about 5 per cent of cases. This incidence is much lower than that of similar changes in the fissula ante fenestram. If such changes are forerunners to the formation of otosclerotic bone, then the observation that otosclerotic bone occurs more commonly anterior than posterior to the stapes has a developmental basis.

TREATMENT OF VASOMOTOR RHINITIS AND ALLIED CONDITIONS WITH SODIUM MORRHUATE

PRELIMINARY REPORT

FRANK E. FISHOF, M.D.
NEW YORK

Vasomotor rhinitis is a pathologic condition of the nose produced by a local or a remote disturbance in the vasomotor mechanism. It is noninfectious in origin. It may, however, occur with an infectious or with an allergic condition of the nose.[1]

There are three types of the disease:[2] (1) acute vasomotor rhinitis; (2) subacute or chronic vasomotor rhinitis, and (3) vasomotor rhinitis, acute or chronic, with osseous hypertrophy. The acute form is characterized by: (*a*) profuse watery discharge; (*b*) pale, edematous, boggy, water-logged mucosa, and (*c*) evanescent obstruction of the nose. (In evanescent obstruction either one or both sides of the nose may be involved. In fact, the congestion may change from one side to the other or may disappear entirely under the examiner's eyes.) In the chronic type there is evidence of repeated attacks of inflammation of the mucosa, manifested by the appearance of polypoid degeneration in different parts of the middle or of the inferior turbinates. The subjective symptoms accompanying this type depend on two factors: (*a*) the extent of polypoid degeneration and (*b*) the extent of congestion of the nonpolypoid turbinates. The cardinal subjective symptoms of vasomotor rhinitis are: (1) obstruction or a sense of fulness in the nose, (2) profuse watery discharge and (3) sneezing. The secondary or referred symptoms may be: (1) pain in the frontal region, (2) heaviness in the head, (3) a bulging sensation in the eyes, (4) noise

---

The sodium morrhuate used was supplied by the Crookes Laboratory, New York.

Read at a meeting of the Clinical Society of the Hospital for Joint Diseases, May 4, 1937.

This work was done at the Hospital for Joint Diseases in the service of Dr. Alfred Braun and the clinic of Dr. Leopold Glushak.

1. Cameron, J. A. M.: Investigation of Part Played by Allergy or Sensitization as Factor in Predisposing Mucous Membrane of Nasal Passages, J. Laryng. & Otol. **50**:493-510 (July) 1935. Cohen, A. I.: Allergy and Its Relationship to Sinusitis and Allied Nasal Conditions, Arch. Otolaryng. **21**:265-271 (March) 1935.

2. Freeman, J.: Paroxysmal Rhinorrhea, J. Laryng. & Otol. **40**:561-567 (Sept.) 1925.

in the ears, (5) pain behind the ears, (6) discharge in the back of the throat, (7) coughing up of phlegm in the morning, (8) a tired feeling, especially in the morning, (9) poor appetite, especially during an attack, and (10) disturbed sleep.

The etiology of vasomotor rhinits is not definitely known. The probable factors producing this condition are: (1) sudden changes in temperature (getting out of a warm bed into a cool room), (2) endocrine disturbances (onset of menses, menopause), (3) disturbances in the calcium-phosphorus ratio and (4) changes in osmotic pressure due to disturbance in the mineral balance of the tissues.

Vasomotor rhinitis should be differentiated from a true allergic or infectious rhinitis.[3] The allergic form is brought on when the patient is exposed to the excitant factor or factors. Thus a person may be sensitive to foods, to dust, to inhalants or to bacterial organisms, and rhinitis may develop when he comes in contact with any of these allergens. In this case, the condition continues as long as the excitant factor is present. If polyps are found the nasal secretion, as well as the contents of the polyps, contains eosinophils. The blood shows an increase in eosinophils. There is little mucin in the secretion, as the goblet cells are impaired in their function. If infectious rhinitis is acute, it is generally associated with fever. If it is chronic, the discharge is mucopurulent and shows many leukocytes and cellular débris; the mucosa is thickened.[4]

As to treatment of vasomotor rhinitis, there are several methods in vogue:

I. Surgical [5]
   (*a*) Turbinectomy
   (*b*) Turbinotomy

II. Cauterization [6]
   (*a*) Chemical—chromic acid bead
   (*b*) Electrical
      1. Unipolar electrode
      2. Bipolar electrode

3. Rusten, E. M.: Diagnosis and Treatment of Vasomotor Rhinitis with Especial References to Allergy, Journal-Lancet **55**:325-329 (May) 1935.

4. Buhrmeister, C.: The Chemical Composition of Secretions from Allergic Rhinitis and Sinus Exudates, Laryngoscope **45**:347-354 (May) 1935.

5. Departout, G.: Surgery of Middle Turbinate in Therapy of Headaches, Presse méd. **43**:1023-1024 (June) 1935. Longhi, A. E., and Genisans, J.: Turbinotomies and Turbinectomy, Rev. méd. del Rosario **24**:555-566 (June) 1934. Dutrow, H. V.: Conservative Surgical Treatment of Hypertrophic Rhinitis, Arch. Otolaryng. **21**:59-63 (Jan.) 1935.

6. Curtis, H.: Abuse of the Electric Cautery in the Nose, Laryngoscope **6**:32 (Jan.) 1899.

III. Phenolization [7]

IV. Ionization (zinc sulfate, sodium chloride) [8]

V. Use of sclerosing chemicals [9]

(*a*) Hypertonic saline solution
(*b*) Sodium salicylate
(*c*) Glycerin
(*d*) Calcium sodium lactate (5 per cent solution)
(*e*) Quinine and urea hydrochloride
(*f*) Alcohol
(*g*) Sodium morrhuate (as proposed by me).

I shall not discuss in detail the objections to the methods other than the use of sodium morrhuate. In general, it may be said that some of these methods produce necrosis of the mucosa and render the ciliated cells inactive. When alcohol is used, it causes degeneration of the nerves. Synechiae may take place between the septum and the turbinates. Too much of the turbinate may be removed or destroyed, and a new disease, namely atrophic rhinitis, may be thus induced.

In 1935 I started to inject sodium morrhuate, after having failed with other methods. The technic applied is as follows:

TECHNIC

The nose is thoroughly cleaned by suction. A 10 per cent solution of cocaine hydrochloride is used to anesthetize and to shrink the mucosa. Cocainization,

7. Palmer, A.: The Effect of Phenol in Hyperesthetic Rhinitis with Tissue Study of the Nasal Mucosa, Ann. Otol. Rhin. & Laryng. **44**:25-32 (March) 1935.

8. Warwick, H. L.: Treatment of Hay Fever and Allied Conditions by Ionization, Laryngoscope **44**:173-181 (March) 1934. Ramiriz, M. A.: Disappointing Results from Ionization, J. A. M. A. **106**:281-282 (Jan. 25) 1936.

9. Monson, L. P.: Treatment of Hypertrophied Inferior Turbinates by Use of Sclerosing Solutions, Arch. Otolaryng. **23**:487-488 (April) 1936. Bontinck, W. J.: Sclerosing Injections in Therapy of Hypertrophied Rhinitis, Rev. de laryng. **54**: 1285-1289 (Dec.) 1933. Ibanez, L. M. G.: Conservative Therapy of Hypertrophied Rhinitis by Sclerosing Injections of Quinine and Urea Hydrochloride, Bol. Asoc. méd. de Puerto Rico **25**:544-549 (Sept.) 1933. Richier: Treatment of Hypertrophied Vasomotor Rhinitis of the Inferior Turbinate by Sclerosing Injections, Otorhino-laryng. internat. **14**:226 (May) 1930. Mahorner, H. R., and Ochsner, A.: Histologic Effects of Intravenous Sclerosing Solution in Subcutaneous Tissues, Arch. Surg. **30**:573-583 (April) 1935. Ochsner, A., and Mahorner, H. R.: Studies of the Comparative Value of Intravenous Sclerosing Substances, Proc. Soc. Exper. Biol. & Med. **30**:1180-1181 (June) 1933. Suárez, A. M.: New Treatment of Hypertrophic Coryza, Rev. méd. latino-am. **19**:53-57 (Oct.) 1933. Calderin, A. M.: Sclerosing Method in Rhinology, Med. ibera **1**:515-517 (April) 1934. Ochsner, A., and Garside, E.: Intravenous Injection of Sclerosing Substances, Ann. Surg. **96**:691-716 (Oct.) 1932. Vail, H.: Treatment of Hay Fever by Nasal Injection of Alcohol, Arch. Otolaryng. **18**:651-657 (Nov.) 1933.

incidentally, aids in disclosing whether or not there is, in addition to the boggy mucosa, an osseous or a polypoid obstruction. By the use of a double curved needle devised by me and a 2 cc. luer-lok or a tuberculin syringe, about 0.25 to 0.5 cc. of a 5 per cent solution of sodium morrhuate is injected between the periosteum of the turbinate and the overlying mucosa. In this way the erectile tissue is infiltrated with the fluid. The needle is inserted in such a manner that it touches the periosteum. Injections may be given at several points of the turbinate on the same occasion. One must be careful not to inject too much of the fluid at one point; nor should the injection be superficial. On withdrawal of the needle there is slight bleeding. This is readily controlled by placing a piece of cotton in the nasal vestibule. From three to five minutes later suction is applied to remove any clot or secretion that may be present. The immediate effect of the injection is increased nasal discharge, difficult breathing through the nose, slight headache and sometimes a sense of fulness in the head. If the solution accidentally drips into the pharynx, the patient complains of a slight burning sensation in the throat and of a fishy odor. This dripping can be prevented by the use of a special needle (the Fishof needle, fig. 1) with proper angulation. The burning sensation in the throat can be neutralized by gargling with a 5 per cent solution of sodium bicarbonate. This

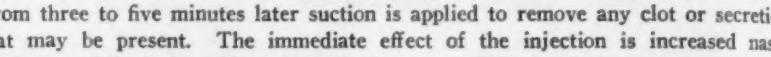


Fig. 1.—The Fishof needle.

after-effect lasts from about twelve to twenty-four hours. Thereafter there is a gradual improvement of both subjective and objective symptoms. On examination at the end of a week, one finds a definite contraction of the mucosa and a great increase in the breathing space. Cocainization of the nose is not necessary when the second or the third injection is given. These injections are given at intervals of one or two weeks. It is important not to give them more often, as the reaction produced may be severe. It is rarely necessary to give more than four injections.

REPORT OF CASES

CASE 1.—A. L., aged 22, complained of sneezing and of watery discharge from the nose. The nose felt stuffed. This condition was of two and a half years' duration. Previous treatment had consisted of cauterization of the anterior tips of the inferior turbinates with chromic acid beads, electric coagulation and injections of bacteriophage in 1935.

Injections of sodium morrhuate were given in 1936 on September 30, October 9 and October 30. The patient observed that after the first injection there was no change. A few days after the second injection, the sneezing decreased and the nasal discharge almost stopped. This relief lasted two weeks, after which time a cold developed. After the third injection, the condition gradually improved and there was no recurrence of the original symptoms.

CASE 2.—D. G., aged 48, complained of pain in the right side of his head, noise in his right ear and a heavy feeling in his head. The condition was of two years' duration. He had had no treatment. The diagnosis was a hyperplastic condition of the inferior turbinates.

On October 14, 0.2 cc. of sodium morrhuate was injected into the right and 0.2 cc. into the left inferior turbinate. On October 21, the same procedure was

repeated. After the first injection, the patient complained of headache and of stuffed nose, which lasted for several hours. The next day she felt better; her breathing improved greatly and the pain ceased. On April 26, 1937, both subjective and objective findings were negative.

CASE 3.—S. E., aged 35, complained of running nose, of sneezing and of difficult breathing. She felt a stuffiness at all times, which alternated from one side of the nose to the other. Most of the time both sides of the nose were involved. Toward evening she felt worse. This condition started in 1936.

The patient had had tests for allergy at another hospital, where she was found to be sensitive to house dust, pyrethrum and rice. She had been given injections of autogenous vaccine from sputum containing Staphylococcus, Streptococcus and Neisseria catarrhalis. She had also been given injections against house dust. She had undergone ionization of the nasal mucosa and had been placed on a calcium-free and salt-free diet. These forms of treatment had had negative results.

On March 10, 0.5 cc. of sodium morrhuate was injected into the right inferior turbinate. Immediately afterward the patient complained of severe pain in the head and of a sense of dizziness. This lasted for several hours. The nasal discharge increased. The next day a definite improvement was noticed; the sneezing was less marked and breathing was easier. On April 14, the same amount of sodium morrhuate was injected into the left inferior turbinate. The patient was told that she could eat anything and was not given any alkali. The results within two weeks were surprisingly satisfactory.

CASE 4.—H. L., aged 32, was troubled with profuse nasal discharge, sneezing, sore throat and occasional head colds. This condition started eight years ago after use of chlorox. The patient had received injections at an otolaryngologic hospital.

Injections of sodium morrhuate were administered on October 28 and on November 4. After the first injection, the patient had a heaviness between the eyes and the bridge of the nose. She sneezed and had a profuse mucoid discharge and an itchy, burning sensation in the back of the throat. On November 4, a few drops of sodium morrhuate was injected into the boggy middle turbinates. Thereafter the patient noticed a definite improvement. She was last examined on April 28, 1937, at which time she was found to be free of nasal obstruction, sneezing and headache.

CASE 5.—F. W., aged 13, had had sneezing and a running nose morning and night since February 1935. Treatment had consisted of cauterization with chromic acid beads and of phenolization. A powder containing calcium had been administered, and feather pillows had been removed. No improvement had been noted.

On July 13, 1936, 0.5 cc. of sodium morrhuate was injected into the right inferior turbinate; on July 29, 0.5 cc. was injected into the left inferior turbinate; on September 21, 0.5 cc. was injected into the right inferior turbinate, and on October 19, 0.6 cc. was injected into the left inferior turbinate.

Immediately after the first injection, the patient had headache lasting several hours and his breathing became more difficult. Later the discomfort gradually subsided. The reactions to subsequent injections were not as marked. On April 26, 1937, the patient stated that he was no longer troubled with nasal disturbances.

CASE 6.—G. B., aged 42, complained of obstructed breathing and pain over the bridge of the nose, rhinorrhea, sneezing and sore throat. The pain was more marked at night. She had to plug the right side of her nose when walking in cold weather or on a windy day to avoid severe pain. This condition started a year ago.

After the first injection of sodium morrhuate, no improvement was noted. On her way home the patient felt dizzy. After the second injection, there was a slight improvement in her breathing. Pain was less marked. After the third and fourth injections, she could sleep better and breathe more easily. The patient had a total of six injections. Results were fair.

CASE 7.—K. A., aged 43, complained of pain and of a throbbing feeling on the left side of her head and face. The right side of her nose felt stuffed, but there

Fig. 2.—Photomicrograph of a section taken from the inferior turbinate a few weeks after injections of sodium morrhuate were given.

was no pain. She also complained of mouth breathing, of lacrimation and of postnasal discharge. This condition started a year ago. Treatment had consisted of cauterization with silver nitrate (10 per cent solution). Examination disclosed vasomotor rhinitis with osseous hypertrophy of the right inferior turbinate.

On July 15, 0.5 cc. of sodium morrhuate was injected into the right inferior turbinate; on July 27, 0.4 cc. was injected into the right inferior turbinate and 0.2 cc. into the left inferior turbinate. After the first injection, the patient felt

uncomfortable for twenty-four hours and had shooting pain in the nose and in the roof of the mouth. There was also profuse nasal discharge. Two days thereafter breathing definitely improved and sneezing subsided.

In view of the osseous hypertrophy, a resection of the turbinate was performed. The microscopic report on the section read: ". . . fragments of nasal mucosa showing subacute inflammation. The epithelium is denuded in places. Some areas show squamous metaplasia." (See photomicrograph, fig. 2.)

SUMMARY

The cases reported are those in which the disease was refractory to other methods of treatment for vasomotor rhinitis but responded favorably to injections of sodium morrhuate. This method is preferred because:

1. It does not destroy the physiologic functions of the nasal mucosa.
2. The amount of contraction of the turbinates can be controlled by regulating the amount of fluid to be injected.
3. By use of the Fishof needle other fluid can be applied at points at which it is most desirable.
4. The drug is inexpensive. An ampule of 5 cc. is more than sufficient for clearing up a condition.
5. The treatment does not require expensive apparatus.

The points to bear in mind are:

1. Not to inject too much fluid at a given point, lest necrosis result.
2. Not to give the injections too often.
3. Not to give an injection in the presence of an acute superimposed infection.

333 East One Hundred and Seventy-Sixth Street.

CONTINUOUSLY OPEN EUSTACHIAN TUBE

GEORGE E. SHAMBAUGH JR., M.D.
CHICAGO

It is not generally known that the continuously open eustachian tube is a definite clinical entity with symptoms which are as distinctive as those produced by pathologic occlusion of the tube. Thus, the continuously open tube has not even been mentioned in such standard textbooks of otology as those of Politzer, Kerrison, Barnhill, Watkyn-Thomas and Yates, Fraser and Turner, and Jackson and Coates. Ballenger devoted a paragraph to the condition but omitted discussion of treatment. Bezold alone gave a complete account of the continuously open tube. The purpose of this communication is to call attention to this generally unrecognized clinical condition, to add several other etiologic factors to the single factor described by Bezold and to add a therapeutic measure to that advised by Bezold.

ANATOMY AND PHYSIOLOGY

The normal eustachian tube lies closed in its cartilaginous portion, forming a vertical slit. This closure is maintained by a thick pad of fat, which surrounds the lateral wall of the cartilaginous tube. During yawning or swallowing the tube opens momentarily, as a result of the action of two muscles, the levator palati and the tensor palati. The former arises in part from the lateral surface of the cartilaginous tube, passes in a tendon around the hamulus of the pterygoid bone and inserts in the hard and the soft palate, so that when it contracts the cartilaginous tube is pulled open. The tensor palati runs along under the cartilaginous tube from the temporal bone to the soft palate, so that when it contracts the tube is pushed on from below and opens.

ETIOLOGY

In Bezold's experience the continuously open eustachian tube occurred only in emaciated patients with advanced tuberculosis or other serious disease, in whom disappearance of the normal pad of fat from around the tube allowed it to fall permanently open. Of four patients with a continuously open eustachian tube observed by me in the past four years, only one was emaciated (case 4). In one patient (case 2) the symptoms began seven weeks after adenoidectomy and were probably due to contraction of scar tissue in the fossa of Rosenmüller. In one

patient (case 3) the condition followed irradiation of a carcinoma of the nasopharynx. The carcinoma disappeared, but part of the torus tubarius had been destroyed, and there was considerable atrophy of the nasal and nasopharyngeal mucosa. In the fourth patient (case 1) the symptoms were associated with simple atrophy of the mucosa of the nose, the nasopharynx and the eustachian orifice.

SYMPTOMS

The clinical symptoms produced by the continuously open eustachian tube are the result of sounds reaching the middle ear directly through the tube from the pharynx. The patient's own voice is heard by him, with an annoying, abnormal loudness (autophonia), and he complains of a roaring sound synchronous with breathing, due to the rush of air back and forth through the upper part of the respiratory tract.

DIAGNOSIS

The diagnosis of continuously open eustachian tube is easily made with an auscultation tube from the patient's ear to the examiner's ear. If the tube is open, the patient's voice is heard clearly through the auscultation tube and has a rough, hollow quality, while his breathing is heard as an intermittent hollow roaring sound. As soon as the tube has been closed by the therapy to be described, the patient's voice and his breathing are no longer heard through the auscultation tube.

TREATMENT

The treatment recommended by Bezold is as simple as it is effective. It consists of insufflation of a little powder composed of 1 part salicylic acid and 4 parts boric acid into the eustachian tube through a eustachian catheter. Salicylic acid is an irritant and causes an inflammatory swelling of the mucosa of the tube which closes it. The patient experiences complete relief from his symptoms within a few minutes, and this relief usually lasts one or two weeks. Since in several of my cases the condition has been associated with simple atrophy of the mucosa of the nose, the nasopharynx and the eustachian orifice, I have added to Bezold's treatment the simple snuffing of physiologic solution of sodium chloride through the nose once or twice a day to stimulate the atrophic mucosa. Two of my four patients have become able to dispense with the insufflations of powdered salicylic and boric acid as a result of this procedure.

REPORT OF CASES

CASE 1.—Mrs. F. S., a slender but not ill nourished housewife of 45, complained of a loud roaring in her ears and hyperacusis to her own voice, which she had

first noted eighteen years before for brief periods during pregnancy but which had become more and more continuous in recent years. Holding the nose closed and sniffing strongly would temporarily stop the symptoms. A head cold gave immediate and lasting relief for its duration. On the other hand, talking, yawning, swallowing and nervousness caused a recurrence of the symptoms. She had consulted several otologists, who had carried out inflations without benefit.

The drum membranes appeared entirely normal. The nasal and nasopharyngeal mucosa was somewhat pale and atrophic, but there was no crusting. The hearing was normal. The eustachian tubes were patent when catheterized, but through the auscultation tube the patient's breathing could be heard as a rough, blowing sound, and her voice came clearly, with a loud, rasping, vibrating quality. When the patient held her nose and sniffed sharply, stopping her symptoms, her breathing and voice could no longer be heard through the auscultation tube. An audiogram was made with the earphone in front of the open mouth and the external auditory canals occluded with wet cotton, after the patient had sniffed and stopped her symptoms (solid line, chart 1), and another was made after she had swallowed and caused a recurrence of her symptoms (dotted line, chart 1). These seemed to prove that her symptoms were due to the fact that the eustachian tubes remained continuously open.

A pinch of powdered salicylic acid and boric acid was blown into each eustachian tube through the eustachian catheter. There was a biting, burning irritation in the nasopharynx, which lasted a few minutes, and the annoying symptoms were instantly relieved. The patient could swallow, talk, yawn or feel nervous to her heart's content for the first time in years without the tubes flying open. To stimulate the pale, atrophic nasal and nasopharyngeal mucosa the patient was instructed to wash the nose with physiologic solution of sodium chloride twice daily. It was necessary to repeat the salicylic-boric acid insufflation every few months during the first year and less often the second year. After that, insufflation was needed only when the patient had been neglectful about irrigating the nasal passages with physiologic solution of sodium chloride. The nasal mucosa has resumed a normal pink, succulent appearance, and for more than two years at the time of this writing no further insufflations of powder have been necessary.

Case 2.—Mr. M. C., a moderately well nourished man of 49, complained of deafness, which had been complete in the left ear and partial in the right ear since a series of attacks of vertigo twenty years before. The left ear showed a genuine attic cholesteatoma, with complete absence of cochlear function but a weak caloric response. The right drum membrane was intact but thinned out and sharply retracted, and hearing tests showed marked obstruction to the conduction of sound. A mass of adenoid tissue in the nasopharynx extended below the upper margin of each choana.

Adenoidectomy was followed by complete and permanent restoration of normal hearing in the right ear (chart 2). Seven weeks later the patient first noted autophonia, which could be relieved by holding the nose and drawing inward but was brought on again by swallowing, especially when the patient was nervous. Insufflation of powdered salicylic and boric acid into the eustachian tube gave complete relief for two days. Because of a moderately developed nasal atrophy, nasal irrigations twice daily with physiologic solution of sodium chloride were advised. With this the symptoms recurred less often, but insufflation every ten days or two weeks was still necessary. The patient's son, a senior medical student, was taught how to carry out this treatment; he has been doing it for two

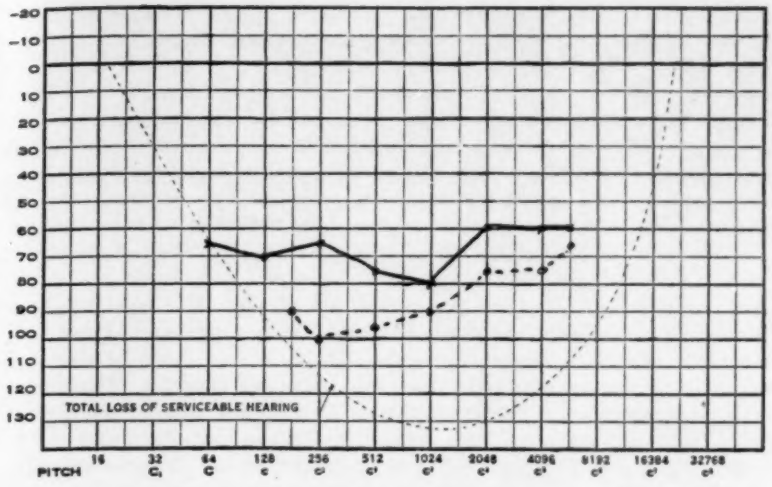


Chart 1 (case 1).—Audiogram taken with the earphone in front of the open mouth and with both external auditory canals occluded with wet cotton. The solid line indicates hearing before the application of therapy, and the dotted one, hearing after the insufflation of powdered salicylic and boric acid in both eustachian tubes.

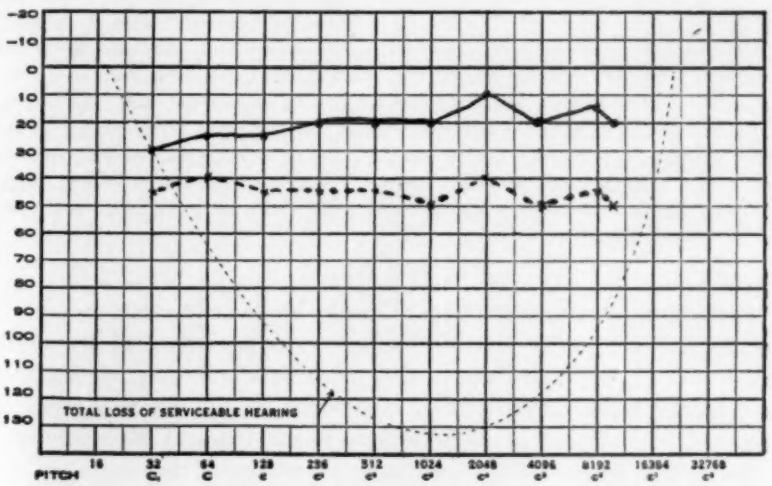


Chart 2 (case 2).—Audiograms of a patient complaining of autophonia. The broken line indicates hearing in the right ear before adenoidectomy, and the solid one, hearing in the right ear after adenoidectomy.

years at the time this report is written. The treatment is just as effective now as when it was first carried out, and it must be repeated about every two weeks.

CASE 3.—G. L. B., a man of 32, complained of increasing soreness in the right side of the throat for three months, deafness and fulness in the right ear for one month and inability to open the mouth more than an inch (2.5 cm.) for one month. Hearing tests showed a marked defect in conduction in the right ear. The right drum membrane was sharply retracted and the left was normal. An irregular mass of tissue filled the upper third of the nasopharynx on the right side. Biopsy showed an undifferentiated type of carcinoma (lympho-epithelioma).

The patient was referred for irradiation with a 4 Gm. radium bomb, and a total of 160,000 milligram hours was given over a period of four weeks, each side of the neck being irradiated twice daily. At the completion of the treatment the tumor mass had disappeared, and all the symptoms had cleared.

Six months later the patient first noticed autophonia in the right ear, brought on by nervous or physical exertion and relieved by holding the nose and sniffing. Examination showed the upper half of the right torus tubarius to be absent. The nasal and nasopharyngeal mucosa was pale, thin and atrophic. The symptoms were immediately relieved by the insufflation of powdered salicylic and boric acid. Nasal irrigations with physiologic solution of sodium chloride caused the nasal atrophy gradually to disappear, and it was necessary to use the powdered salicylic and boric acid only once again. The patient had remained free from any symptoms for more than two years, and the nasal mucosa appeared entirely normal when this report was made.

CASE 4.—Miss R. G., aged 39, was first seen by me toward the termination of a long illness due to inoperable carcinoma of the ovary. She complained of blowing in her ears. She was extremely emaciated, having lost more than half her previous weight in several months. I found the drum membranes normal, the eustachian tubes patent when catheterized and the hearing normal. The nasal mucosa was pale, thin and atrophic. I could offer no explanation or treatment for the tinnitus. A week later I was asked to see her again, because of the great annoyance from the blowing in her ears, and again I could suggest no cause or treatment. The following day, still puzzled, I reexamined the ears, and this time the patient remarked that the extreme loudness of her voice bothered her as much as the roaring. Questioning her, I learned that the roaring was synchronous with respiration. The diagnosis was then apparent, and insufflation of powdered salicylic and boric acid gave immediate and complete relief from the symptoms. The patient died of inanition four days later.

CONCLUSION

Continuously open eustachian tube may be caused by extreme emaciation, as pointed out by Bezold, owing to disappearance of the normal pad of fat from around the cartilaginous tube. It may, on the other hand, be the result of atrophy of the mucosa in the mouth of the tube, of contraction of scar tissue adhesions in the fossa of Rosenmüller or of destruction of part of the cartilaginous orifice of the eustachian tube by carcinoma.

In every case of low-pitched tinnitus of obscure cause, continuously open eustachian tube should be considered. The concomitant symptom of autophonia and the frequency with which relief can be obtained by

holding the nose and sniffing strongly further suggest the condition. The diagnosis may be made by hearing the patient's breathing through the auscultation tube and by the therapeutic test of insufflation of powdered salicylic and boric acid. The treatment is simple and effective, consisting of occasional insufflation of a pinch of powder made up of 1 part salicylic acid and 4 parts boric acid plus treatment of any associated atrophy of the nasal and nasopharyngeal mucosa by nasal irrigations with physiologic solution of sodium chloride once or twice daily.

122 South Michigan Avenue.

INFLAMMATORY BRONCHIAL TUMORS

REPORT OF A CASE AND REVIEW OF THE LITERATURE

BERTHOLD S. POLLAK, M.D.
SECAUCUS, N. J.
AND
SAMUEL COHEN, M.D.
AND
ANGELO M. GNASSI, M.D.
JERSEY CITY, N. J.

Within the past decade there has been sustained interest in the study of benign bronchial growths. The clinical stimuli have been afforded by (1) greater use of the bronchoscope in pulmonary conditions; (2) the urgent desire for effective therapeusis; (3) careful postmortem examination, and (4) increasing knowledge of the mechanical and biologic sequelae of these tumors. In this paper we shall present the striking clinical and pathologic protocols of a case of inflammatory bronchial polyp in an elderly man who came to postmortem examination. A review of the literature on primary benign bronchial tumors and inflammatory tumor-like masses is appended.

REPORT OF CASE

Clinical Protocol.—History: C. F., a white man aged 55, was admitted to the tuberculosis service on Dec. 9, 1935. He had had a cough at intermittent intervals for four years. This had become constant during the preceding year and at the time of admission was productive of 4 ounces (118 cc.) of nonfoul mucopurulent sputum. There was no hemoptysis. Occasionally during the preceding twelve months there had been pain in the thorax and the upper part of the abdomen, accompanied with vomiting, constipation for six months, fatigability and loss of weight (quantity unknown).

Physical Examination: At the time of admission the patient was pale and thin and appeared to be chronically ill. The right pupil was slightly larger than the left, but both reacted to light and in accommodation. The trachea was deviated to the right. The chest was emphysematous and some dilatation of the superficial veins was present over the upper anterior aspect of the thorax and the right arm and to a lesser degree on the left arm. There was dulness in the right lung from the apex anteriorly to the fourth rib, in the apex of the axilla and posteriorly from the apex to the sixth vertebral spine. Bronchial breathing was heard in these areas, and there were a few rales posteriorly. The left lung disclosed hyperresonance throughout. The heart and the abdomen showed no abnormality.

From the Tuberculosis Service and the Department of Pathology of the Medical Center of Jersey City.

In the extremities thickening of the walls of the peripheral vessels was noted, and there were cyanosis of the nails and slight clubbing of the fingers. Rectal examination showed the prostate to be slightly enlarged.

Laboratory Data: The blood count at the time of admission showed 3,120,000 red blood corpuscles, 70 per cent hemoglobin and 11,350 white blood corpuscles, with 80 per cent polymorphonuclear leukocytes and 20 per cent lymphocytes. The nonprotein nitrogen and the sugar content of the blood were within normal limits. The Wassermann reaction was negative. Urinalysis showed no abnormality. Ten tests of concentrated sputum (seventy-two hour specimens) showed no sign of tubercle bacilli, and a guinea pig inoculated with the sputum showed no evidence

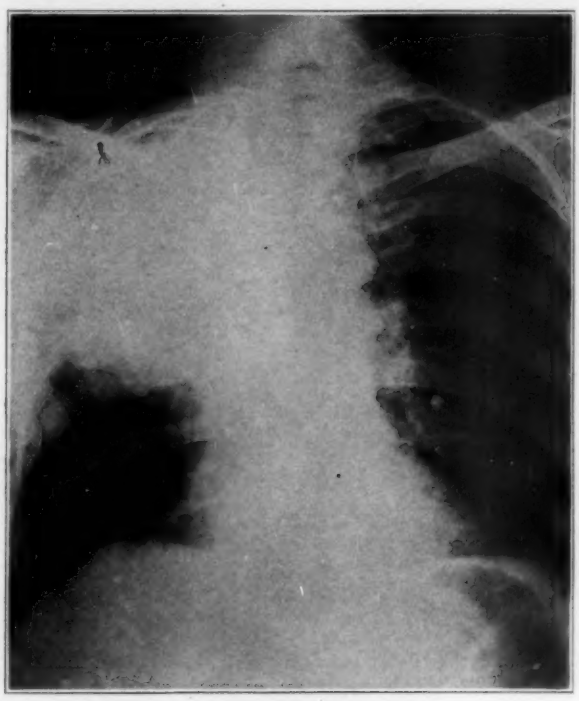


Fig. 1.—Roentgenogram of the chest, showing homogeneous density from the apex to the fourth anterior rib (right), with strandlike infiltration in the remainder of the pulmonary field. The right side of the diaphragm is irregular. There is a calcified focus in the apex of the left lung, with a generalized increase in linear markings. The trachea is displaced to the right.

of tuberculosis at autopsy. A roentgenogram of the chest on Dec. 13, 1935 (fig. 1) revealed a homogeneous density extending from the apex to the fourth anterior rib (right), with strandlike infiltration in the remainder of the pulmonary field. The right side of the diaphragm was peaked at its midpoint. The left lung showed a calcified focus in the apex and increased pulmonary markings. The trachea was deviated to the right; the cardiac shadow showed no abnormality.

Clinical Course: Because of persistent constipation a barium enema was given, and examination showed marked dilatation of the cecum and the colon but no evidence of organic obstruction. Proctoscopic examination showed no abnormality.

On Dec. 18, 1935, the patient's temperature rose to 105 F., and physical signs revealed, in addition to the original findings, bronchopneumonia and acute pleuritis of the lower lobe of the right lung. The temperature ranged between 100 and 104 F. for the next two weeks and thereafter between 98 and 101.5 F. on the average, with an occasional elevation to 102 or 103 F.

Two bronchoscopic examinations, on Dec. 31, 1935, and Feb. 14, 1936, were reported as showing no evidence of intratracheal or intrabronchial erosion or

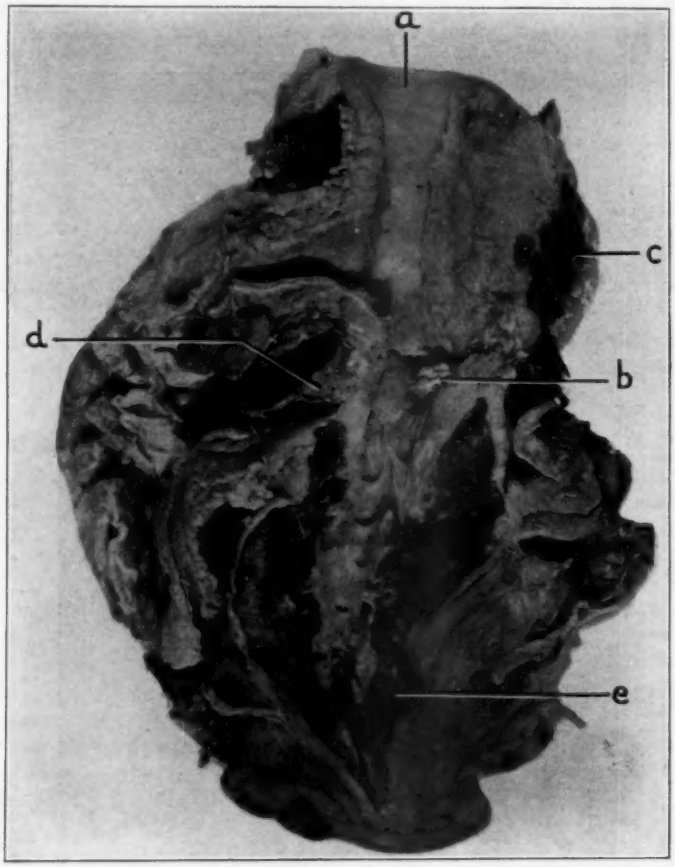


Fig. 2.—Section through the trachea and the bronchi, showing *a*, the trachea; *b*, an inflammatory polyp at the mouth of the bronchus of the right upper lobe; *c*, a portion of the right upper lobe; *d*, enlarged anthracotic infratracheal glands, and *e*, the right lower lobe.

growth. A thick mucopurulent secretion was noted in the right main bronchus. Postural drainage was advised but not tolerated by the patient. The findings of a third bronchoscopic examination, on April 28, 1936, were as follows: The mouth of the bronchus of the upper lobe of the right lung was almost completely obstructed by a somewhat irregular mass of tissue. Histologic examination showed necrotic and inflammatory changes. The patient continued to expectorate 4 or 5

ounces (from 118 to 148 cc.) of slightly foul thick sputum. A roentgenogram taken in March showed a few areas of rarefaction within the density of the upper part of the right pulmonary field. In April the liver was palpable just below the costal margin. No superficial adenopathy was found. A loss of 7 pounds (3.2 Kg.) occurred between December 1935 and May 1936. The patient's general condition became progressively worse, and he died in a state of asthenia and respiratory decompensation on May 14.

The final clinical diagnosis was bronchial carcinoma.

Fig. 3.—Diffusely inflamed tissue covered by transitional epithelium (× 45).

Pathologic Protocol (Pertinent Observations).—Grossly, both pleural cavities were obliterated by dense fibrous adhesions, the right more so than the left. A portion of the upper lobe of the right lung was torn in the process of freeing it from the thoracic cage. When the trachea and the bronchi were opened there was noted, about 1.5 cm. below the carina, on the anterolateral wall of the main bronchus, a pedunculated growth the base of which measured 1.5 cm. in diameter and which almost completely occluded the orifice of the bronchus of the right upper lobe (fig. 2). The base of the mass was firm and grayish, while the free surface was soft and friable. The bronchial mucosa lined the attached portion and lower edges of the mass. The main bronchus was reddened and contained a thick mucopurulent secretion. The upper lobe, firm, reduced in volume and resilient, with

a diffuse bluish black pigmentation, revealed multiple irregular necrotic areas. The mouths of some bronchi were dilated, and these exuded rather foul thick secretions. The middle lobe showed marked anthracosis and a few small grayish patches. The lower lobe revealed anthracosis, considerable honeycombing of the cut surfaces and a few scattered, noncrepitant, grayish granular patches of lobular distribution. In the left lung the oblique fissure was obliterated by fibrous bands. The grossly normal bronchi contained a moderate quantity of frothy mucus. The parenchyma of the lung showed widespread anthracosis, a few scattered strands of fibrosis and a dense white calcified lesion measuring about 0.5 cm. in the posterior apical portion of the upper lobe. A honeycombed appearance was noted in the inferior posterior portion of the lower lobe.

The peribronchial and infratracheal glands were enlarged, fairly firm and bluish black, with numerous whitish strands. No evidence of malignant change was observed.

Microscopic section through the bronchial growth (fig. 3) showed strands or bundles of fibrous tissue permeated by collections of lymphocytes, monocytes and plasma cells and moderately sprinkled with polymorphonuclear cells. There was considerable intracellular and extracellular edema. The blood vessels were well formed but congested and dilated. Areas of myxomatous tissue were infrequent. The covering epithelium was well differentiated, pseudocolumnar, abruptly eroded in places and replaced by inflammatory cells.

The final anatomic diagnosis showed an inflammatory bronchial polyp, anthracosis, chronic purulent bronchitis, bronchiectasis, pulmonary fibrosis and emphysema, chronic suppurative pneumonitis, fibrosis of the pleura and old calcified tubercle.

COMMENT AND REVIEW OF LITERATURE

A clinical diagnosis of bronchogenic carcinoma was made, because this was considered most likely for a man at the age of greatest susceptibility to cancer who presented the manifestations of pulmonary suppuration. Roentgenograms of the chest showed a characteristic picture. The fact that histologic examination of a biopsy specimen did not confirm the diagnosis did not necessarily exclude underlying carcinoma, for it is well known that it is not unusual to obtain with a punch a piece of collateral inflammatory tissue in cases in which the condition is later proved to be cancer. The difficulty of getting "around the corner" for bronchoscopic visualization of growths in the bronchus of the upper lobe of the right lung is also common. The value of repeated examinations is illustrated.

The postmortem discovery of a benign bronchial "tumor" was unexpected. Yet, in a sense, this mass was clinically malignant, since if considered primary it caused suppurative changes distal to the occluded bronchus and if considered secondary to the pulmonary process it certainly aggravated these changes. The sequence of events cannot be definitely proved one way or the other. The clinical and the pathologic data emphasized the hazard of aspiration-induced bronchopneumonic lesions from the primary involved lobe into dependent portions of the same or the opposite lung. Thus, the importance of early recognition

and extirpation of a benign growth before extensive pathologic reactions have occurred is apparent. Another cogent reason for early diagnosis and treatment is that a benign tumor may undergo malignant change. Our patient at the time of admission was debilitated and had advanced

TABLE 1.—*Primary Benign Bronchial Tumors and Inflammatory "Tumors" Diagnosed at Autopsy (Total, 27 Cases)*

Date	Author	Sex of Patient	Age	Type of Tumor	Location of Tumor
1861	von Rokitansky	F	72	Submucous lipoma	Bronchus of left lower lobe
1882	Mueller	F	22	Pedunculated adenoma with partial calcification	Left main bronchus
1883	Chiari	F	68	Mixed tumor of lipomatous, adenomatous and chondromatous tissue	Bronchus of left lower lobe
		M	73	Adenoma	Bronchus of right lower lobe
1888	Radestock	F	21	Colloid struma arising from an aberrant thyroid	Right main bronchus
1892	Siegert	F	61	Chondroma	Bronchus of right middle lobe
1910	Blecher	F	21	Ossified chondroma	Left main bronchus near carina
1916	Heck	F	32	Polyp	Right main bronchus
1917	Kirch	M	44	Enchondromatous polyp	Left main bronchus
1918	Knack	F	47	Pedunculated fibroma	Bronchus of right lower lobe
1922	Feller	F	66	Lipoma	Bronchus of right middle lobe
1925	Caussade and others	M	53	Enchondroma	Right main bronchus
1927	Heine	F	44	Polypoid adenoma	Bronchus of right middle lobe
1927	Reisner	F	47	Polypoid adenoma	Bronchus of right lower lobe
1930	Paul	M	60	Polypoid myxochondroma	Left main bronchus
1931	Geipel	F	33	Adenomatous polyp	Bronchus of right upper lobe
		F	50	Adenomatous polyp	Left main bronchus
1932	Wessler and Rabin	F	32	Lymphoma	Bronchus of right lower lobe
		M	48	Adenoma	Bronchus of left lower lobe
		M	35	Adenoma	Bronchus of right lower lobe
		F	47	Adenomatous polyp	Bronchus of left lower lobe
1932	Moore	M	68	Polypoid chondroma	Bronchus of right lower lobe
1932	Alstead	M	68	Submucous leiomyoma (pedunculated)	Right main bronchus near carina
1934	Honig	M	54	Lipomas	Left main bronchus
1934	Fried	M	57	Adenoma	Left main bronchus
1936	Cabot cases*	F	67	Adenoma	Bronchus of right lower lobe
		M	19	Adenoma	Bronchus of right lower lobe

* Eight cases of adenoma (Peterson, H. O.: Am. J. Roentgenol. **36**: 896 [Dec.] 1936) were mentioned in the report of the Cabot cases.

pulmonary suppuration. It is doubtful whether appropriate treatment would have effected a cure if a correct diagnosis of a benign bronchial process had been made.

The growth observed at autopsy was not an actual neoplasm from a histologic point of view but a tumor-like inflammatory hyperplastic polyp. However, as the Jacksons suggested in 1932, the microscopic

TABLE 2.—*Primary Benign Bronchial Tumors and Inflammatory Bronchial "Tumors" Diagnosed by Bronchoscopy and by Biopsy (Total, 77 Cases)*

Date	Author	Sex of Patient	Age	Type of Tumor	Location of Tumor	Method of Treatment
1907	von Eicken (1 case)	M	41	Enchondroma	Left main bronchus	Bronchoscopy
1910	Speiss (1 case)	M	47	Polyp	Right main bronchus	Bronchoscopy
1915	Jackson (1 case)	M	16	Fibroma	Bronchus of left upper lobe	Bronchoscopy
1920	Pfeiffer (1 case)	F	27	Fibroma	Left main bronchus	Bronchoscopy
1923	Wessler and Jaches (1 case)	M	28	Fibroma	Left main bronchus	Bronchoscopy
1926	Jesberg (1 case)	M	6	Polyp	Left main bronchus	Bronchoscopy
1927	Kernan (1 case)	M	50	Lipoma	Right main bronchus near carina	Bronchoscopy
1927	Zinn (1 case)	M	45	Fibroma	Left main bronchus	Bronchoscopy
1927	Syme (1 case)	F	42	Papillomas	Both main bronchi	Bronchoscopy
1927	Burrell and Trail (1 case)	F	23	Fibroma	Bronchus of left lower lobe	Bronchoscopy
1928	Myerson (1 case)	M	47	Fibrolipoma	Left main bronchus	Bronchoscopy
1929	Lemann (1 case)	F	26	Multiple polyps	Right main bronchus	Bronchoscopy
1929	Ashbury (1 case)	M	40	Fibroma	Left main bronchus	Bronchoscopy
1929	Yankauer (1 case)	M	30	Polyp	Right main bronchus	Bronchoscopy
1929	Hajek (1 case)	M	74	Fibroma	Both bronchi	Bronchoscopy
1929	Aucoin (1 case)	F	39	Pedunculated granuloma	Right main bronchus	Bronchoscopy
1930	Kramer (2 cases)	F	36	Adenoma	Just below orifice of right middle lobe	Bronchoscopy
		M	26	Adenoma	Bronchus of left lower lobe	Bronchoscopy
1931	Zamora (1 case)	M	24	Multiple papillomas*	Larynx, trachea and left main bronchus	Bronchoscopy
1932	Orton (1 case)	M	3	Papillomas*	Larynx and left main bronchus	Bronchoscopy
1932	Jackson and Jackson (8 cases)	F	64	Lipoma	Left main bronchus	Bronchoscopy
		M	46	Multiple papillomas*	Trachea and right main bronchus	Bronchoscopy
		M	63	Inflammatory polyp	Right main bronchus	Bronchoscopy
		M	64	Multiple "osteomas"*	Trachea and right main bronchus	Bronchoscopy
		F	32	Adenomatous polyp	Bronchus of left lower lobe	Bronchoscopy
		F	25	Nonspecific inflammatory granuloma	Bronchus of right lower lobe	Bronchoscopy
		F	15	Nonspecific inflammatory granuloma	Bronchus of right lower lobe	Bronchoscopy
		M	41	Enchondroma	Bronchus of right lower lobe	Bronchoscopy
1932	Wessler and Rabin (13 cases)	M	28	Adenoma	Left main bronchus	Bronchoscopy
		F	36	Adenoma	Bronchus of right middle lobe	Bronchoscopy
		F	17	Adenoma	Bronchus on right lower lobe	Bronchoscopy (not entirely successful because of bleeding)
		M	26	Adenoma	Bronchus of left lower lobe	Bronchoscopy and insertion of radon seeds
		M	38	Inflammatory polyp	Bronchus of right middle lobe	Bronchoscopy

* These tumors were probably not primary in the bronchus (transplants ?).

TABLE 2.—*Primary Benign Bronchial Tumors and Inflammatory Bronchial "Tumors" Diagnosed by Bronchoscopy and by Biopsy (Total, 77 Cases)—Continued*

Date	Author	Sex of Patient	Age	Type of Tumor	Location of Tumor	Method of Treatment
		M	48	Pedunculated tumor (histologic picture ?)	Bronchus of left lower lobe	Bronchoscopy
		F	27	Adenoma	Left main bronchus	Patient refused treatment
		M	64	Pedunculated tumor (benign grossly; no histologic diagnosis)	Left main bronchus near orifice of bronchus of left upper lobe	Tumor not removed; patient unimproved
		M	36	Inflamed polyp	Bronchus of right lower lobe	Bronchoscopy
		F	54	Benign tumor (histologic picture ?)	Left main bronchus	No mention
		F	44	Adenoma	Right main bronchus	No mention
		M	32	Adenoma	Right main bronchus	Removed by electrocautery through bronchoscopy
		F	35	Adenoma	Bronchus of right lower lobe	No mention
1933	Turner (2 cases)	M	25	Multiple polypoid masses	Bronchus of right lower lobe	Bronchoscopy and topical application of silver nitrate
		F	24	Polypoid masses	Left main bronchus	Bronchoscopy and instillation of mono-p-chloro phenol
1934	Beutel (1 case)	M	21	Multiple papillomas*	Trachea and both main bronchi	Bronchoscopy
1935	Miller (1 case)	F	23	Polyp	Bronchus of left upper lobe	Bronchoscopy
1935	Kernan (10 cases)	..	..	Adenoma of bronchus	.....	Lobectomy in 2, spontaneous recovery in 1, roentgen treatment in 1, diathermy in 6, with insertion of radon seeds in addition in 2
1935	Morlock and Pinchin (9 cases)	M	58	Hemangioendothelioma	At level of bronchus of right middle lobe	.....
		M	58	Inflammatory polyp	Bronchus of left upper lobe	Insertion of radon seeds
		F	22	Hemangioendothelioma	Bronchus of right middle lobe	Insertion of radon seeds and diathermy
		F	53	Originally diagnosed as carcinoma; benign tumor, probably adenomatous; patient lived 3 years afterward	Bronchus of right lower lobe	Insertion of radon seeds
		M	45	Benign tumor (originally diagnosed as carcinoma) probably an adenoma; patient lived 2½ years afterward	Bronchus of right middle lobe	Insertion of radon seeds
		M	45	Inflammatory polyp	Bronchus of left lower lobe	Bronchoscopy
		F	21	Hemangioendothelioma	Bronchus of right upper lobe	Insertion of radon seeds and diathermy
		F	41	Granulomatous polyp	Bronchus of right lower lobe	Bronchoscopy
		F	30	Originally diagnosed as possible carcinoma; probably adenoma, since patient was living 5 years later	Bronchus of left lower lobe	Insertion of radon seeds and diathermy
1935	Rosenblum and Klein (1 case)	M	11	Adenomatous polyp	Right main bronchus	Bronchoscopy

TABLE 2.—*Primary Benign Bronchial Tumors and Inflammatory Bronchial "Tumors" Diagnosed by Bronchoscopy and by Biopsy (Total, 77 Cases)—Continued*

Date	Author	Sex of Patient	Age	Type of Tumor	Location of Tumor	Method of Treatment
1935	Kramer and Som† (4 cases)	..	..	Adenoma	.....	.....
		(No detailed description of cases)				
1936	Cabot‡ (6 cases)	..	..	Benign adenoma	.....	Surgical procedure in 4, insertion of radon seeds in 1, insertion of radon seeds and roentgen irradiation in 1
1936	Nienhuis (1 case)	F	46	Inflammatory polyp	Right middle lobe	Bronchoscopy plus topical application of silver nitrate (the polyp was reputed to have followed drainage of an empyema into the middle lower bronchus)
1936	King and Som (1 case)	M	30	Adenoma	Bronchus of right lower lobe	Bronchoscopy and diathermy
1936	Gerlings (1 case)	F	39	Adenoma	Left main bronchus	Bronchoscopy

† Kramer and Som referred to 23 cases of adenoma encountered at the Mount Sinai Hospital, but 19 of these had been reported elsewhere (by Kramer in 1930 and by Wessler and Rabin in 1932).

‡ Eight cases of adenoma (Peterson, H. O.: Am. J. Roentgenol. **36**: 836 [Dec.] 1936) were mentioned in the report of the Cabot cases.

TABLE 3.—*List of Growths, Arranged in Order of Frequency of Occurrence (Recapitulation of Data in Tables 1 and 2 [Total, 104 Cases])*

Type of Growth	No. of Cases	Type of Growth	No. of Cases
Adenoma (including hemangioendothelioma)	51	Mixed tumors	4
Inflammatory "tumor" (including simple polyp, papilloma and granuloma)	23	Fibrolipoma 1 Myxochondroma 1 Adenomatous, myxomatous, chondromatous tumor 1 Ossified chondroma 1	
Fibroma	8	Unclassified tumors	3
Chondroma	6	Leiomyoma	1
Lipoma	5	Lymphoma	1
		Osteoma	1
		Colloid struma arising from aberrant thyroid gland	1

TABLE 4.—*Inflammatory Hyperplastic Tumor-Like Growths (Total, 23 Cases)*

Sex	Age, Years							Total
	1-9	10-19	20-29	30-39	40-49	50-59	60-69	
Male	2	0	3	3	3	1	1	13
Female	0	1	4	2	3	0	0	10
Total	2	1	7	5	6	1	1	23

TABLE 5.—*Sites of Inflammatory Growths (Total, 23 Cases)*

Site	No. of Growths	Site	No. of Growths
Right main bronchus	6	Both main bronchi	1
Bronchus of right lower lobe	5	Trachea and both main bronchi	1
Bronchus of right middle lobe	2	Trachea and right main bronchus	1
Left main bronchus	2	Larynx, trachea and left main bronchus	1
Bronchus of left upper lobe	2	Larynx and left main bronchus	1
Bronchus of left lower lobe	1		

picture is not always well defined and such an inflammatory "tumor" may be the forerunner of a true neoplasm, benign or malignant.

In tables 1, 2 and 3 we have compiled, after a careful search of the literature up to 1937, 104 recorded cases of primary benign tumor or inflammatory hyperplastic tumor-like formation of the bronchial tree, diagnosed at autopsy and microscopic study of tissue removed through the bronchoscope. In 5 cases, designated by asterisks in table 2, the growth probably was not primary (transplant?). Variations in histologic nomenclature must be considered in evaluating these cases for any statistical presentation. We believe that these growths probably occur more commonly than the tabulation would indicate. For example, one need only refer to the influx in recent literature of lesions described as benign adenoma (often diagnosed as carcinoma heretofore) to visualize what more common recourse to bronchoscopy and reconsideration of clinical and pathologic criteria have accomplished to revise the accepted views concerning the incidence and behavior of these tumors.

Of 23 inflammatory hyperplastic growths (including those listed as simple polyps, papillomas and nonspecific granulomas), 22 were found by bronchoscopic examination and 1 at autopsy. They represent 22 per cent of the listed tumors, and their incidence is exceeded only by that of adenomas (49 per cent). The former percentage may be too low, for some writers might include other described lesions in the inflammatory group. The distribution among the various age groups in both sexes and according to location in the bronchi is shown in tables 4 and 5. Note particularly that 8, or 35 per cent, were found in persons of 40 or more years of age—the age of greatest susceptibility to cancer.

According to Peroni, these "tumors" should not be confused with simple endobronchial granulation tissue seen in pyogenic pulmonary processes. He wrote:

> Granulation tissue depending on the irritating action produced by the traumatism of coughing and by the toxic action of pus comes from a bare stroma and is not covered by epithelium, while the inflammatory tumors lift the bronchial epithelium en masse and only in a second stage undergo a metaplastic transformation.
>
> In the latter cases, the main stroma is predominantly of the connective tissue type, with a fibrous myxomatous tendency or characterized by vascular enlargements. In this connective tissue there is almost no sign of what may be the inflammatory origin of the tumor. There are no signs of regression or involution. The entire tumor has a permanent character.
>
> These granulations, which are definitely of irritative origin, have a much more labile character than that of the inflammatory tumors; they are soft, flat and bloody, and as easily as they are produced they disappear by spontaneous resorption or by necrosis. As they are always flat, multiple and sessile, they cover the bronchial wall; they seldom produce phenomena of bronchial obstruction.

The origin of the inflammatory growths is still a moot question. Are they primary or secondary to suppurative pulmonary conditions?

The Jacksons have leaned toward the latter hypothesis, in the belief that infectious bronchial exudate is one of the main causative factors. Peroni expressed doubt of this, for in that case they would be seen more frequently in patients with bronchiectasis and abscess formation. Furthermore, he was unable to reproduce them experimentally in dogs after prolonged irritation of the bronchial mucosa by various agents.

The serious clinical and pathologic sequelae, however, in cases in which such tumors have remained untreated are universally recognized. They constitute a challenge for early diagnosis and removal.

SUMMARY

We have reported the case of a person at the age of highest susceptibility to cancer who suffered from a growth which was erroneously diagnosed clinically as bronchial carcinoma and was discovered at autopsy to be an inflammatory benign bronchial polyp. The outstanding pathologic changes were those of chronic suppurative pneumonitis, bronchiectasis, fibrosis and emphysema. Whether the polyp was primary or secondary could not be definitely determined. The diagnostic pitfalls are mentioned.

From the literature up to 1937, 104 cases of primary benign bronchial tumor and inflammatory tumor-like mass have been reported; these are classified in convenient form. The incidence of inflammatory tumor-like masses, according to our compilation, is 22 per cent, ranking second to that of adenomas. The cause of these growths has not been settled. Clinical alertness is necessary for their early diagnosis and removal.

The bronchoscopic examinations in our case were done by Dr. Milton G. Borrone, Chief of the Oto-Rhino-Laryngological Department of the Medical Center of Jersey City.

BIBLIOGRAPHY

Alstead, S.: Lancet **2**:339 (Aug. 13) 1932.

Ashbury, H. E.: Am. J. Roentgenol. **21**:452 (May) 1919.

Aucoin, E.: Asthmatic Symptoms Due to Granulomas of Both Main Bronchial Orifices Complicated by Cicatrical Stenosis of the Left Main Bronchus, Arch. Otolaryng. **9**:362 (March) 1929.

Beutel, A.: Roentgenpraxis **6**:287 (May) 1934.

Blecher: Mitt. a. d. Grenzgeb. d. Med. u. Chir. **21**:337, 1910.

Burrell, L. J. S., and Trail, R. R.: Lancet **2**:1180 (Dec. 3) 1927.

Cabot Cases 22231 and 22232, New England J. Med. **214**:1149 (June 4) 1936.

Caussade, G., and others: Bull. et mém. Soc. méd. d. hôp. de Paris **49**:1299, 1925.

Chiari: Prag. med. Wchnschr. **51**:497, 1883.

Feller, A.: Virchows Arch. f. path. Anat. **236**:470, 1922.

Fried, B. M.: Adenoma of Bronchial Mucous Glands, Arch. Otolaryng. **20**:375 (Sept.) 1934.

Geipel, P.: Frankfurt. Ztschr. f. Path. **42**:516, 1931.

Gerlings, P. G.: J. Laryng. & Otol. **51**:508 (Aug.) 1936.

Hajek, M.: Laryngoscope **39**:75 (Feb.) 1929.

Heck, W.: Ueber primäres polypöses Bronchialkarzinom mit einer Tafel, Inaug. Dissert., Bonn, H. Trapp, 1916.
Heine, J.: Verhandl. d. deutsch. path. Gesellsch. **22**:293, 1927.
Honig, A.: Monatschr. f. Ohrenh. **68**:155 (Feb.) 1932.
Jackson, C.: Peroral Endoscopy and Laryngeal Surgery, St. Louis, Laryngoscope Co., 1915, p. 432.
——and Jackson, C. L.: Benign Tumors of Trachea and Bronchi, with Especial Reference to Tumor-Like Formations of Inflammatory Origin, J. A. M. A. **99**:1747 (Nov. 19) 1932.
Jesberg, S.: Ann. Otol., Rhin. & Laryng. **35**:905 (Sept.) 1926.
Kernan, J. D.: Laryngoscope **37**:62 (Jan.) 1927; **45**:760 (Oct.) 1935.
King, F. H., and Som, M. J.: J. Mt. Sinai Hosp. **3**:79 (July-Aug.) 1936.
Kirch, E.: Centralbl. f. allg. Path. u. path. Anat. **28**:545, 1917.
Knack: Deutsche med. Wchnschr. **44**:1007, 1917.
Kramer, R.: Ann. Otol., Rhin. & Laryng. **39**:689 (Dec.) 1930.
——and Som, L. M.: ibid. **44**:861 (Sept.) 1935.
Lemann, I. I.: M. Clin. North America **12**:1329 (March) 1929.
Miller, J. W.: Noncancerous Epithelial Tumor Obstructing Bronchus of Upper Lobe of the Left Lung, Arch. Otolaryng. **21**:703 (June) 1935.
Moore, R. A.: Centralbl. f. allg. Path. u. path. Anat. **55**:321 (Sept. 30) 1932.
Morlock, H. V., and Pinchin, A. J. S.: Brit. M. J. **2**:332 (Aug. 24) 1935.
Mueller, J.: Inaug. Dissert., Halle, 1882, p. 15; cited by Patterson.
Myerson, M. D.: Am. J. M. Sc. **176**:720 (Nov.) 1928.
Nienhuis, J.: Nederl. tijdschr. v. geneesk. **80**:4526 (Oct. 3) 1936.
Orton, H. B.: Ann. Otol., Rhin. & Laryng. **41**:933 (Sept.) 1932.
Patterson, E. J.: Benign Bronchial Neoplasms: Bronchoscopic Aspects, Arch. Otolaryng. **12**:739 (Dec.) 1930.
Paul, F.: Monatschr. f. Ohrenh. **64**:669 (June) 1930.
Peroni, A.: Inflammatory Tumors of Bronchi, Arch. Otolaryng. **19**:1 (Jan.) 1934.
Pfeiffer, W.: Deutsche med. Wchnschr. **46**:1298, 1920.
Radestock: Beitr. z. path. Anat. u. z. allg. Path. **3**:289, 1888.
Reisner, D.: Intrabronchial Polypoid Adenoma, Arch. Surg. **16**:1201 (June) 1928.
Rosenblum, P., and Klein, R. I.: J. Pediat. **7**:791 (Dec.) 1935.
Siegert, F.: Virchows Arch. f. path. Anat. **129**:413, 1892.
Speiss, G.: München. med. Wchnschr. **57**:2095, 1910.
Syme, W. S.: J. Laryng. & Otol. **42**:752 (Nov.) 1927.
Turner, P. A.: Kentucky M. J. **31**:423 (Sept.) 1933.
von Eicken, C.: Verhandl. d. Ver. süddeutsch. Laryng., 1907, p. 410.
von Rokitansky, C.: Lehrbuch der pathologischen Anatomie, Vienna, W. Braumüller, 1861, vol. 5, p. 25.
Wessler, H., and Jaches, L.: Clinical Roentgenology of Diseases of the Chest, Troy, N. Y., The Southworth Company, 1923, p. 51.
——and Rabin, C. B.: Am. J. M. Sc. **183**:164 (Feb.) 1932.
Yankauer, S.: Laryngoscope **39**:549 (Aug.) 1929.
Zamora, A. M.: J. Laryng. & Otol. **46**:829 (Dec.) 1931.
Zinn, W. F.: Ann. Otol., Rhin. & Laryng. **36**:278 (March) 1927.

EFFECTS OF DRUGS ON THE STRIA VASCULARIS

W. P. COVELL, M.D.
SAN FRANCISCO

The clinical literature dealing with drugs known to cause impairment of hearing has recently been reviewed by Taylor.[1] The length of the list of substances affecting hearing is as appalling as the diversity of their natures. An experimental approach to the problem will prove fruitless unless studies on a few of these agents are made from various angles. Histologic changes in the peripheral auditory mechanism, as well as lesions which might be apparent in the central auditory pathways, when correlated with the results of hearing tests on animals should finally aid in a better understanding of the action of such drugs as quinine. Once it is possible to find and evaluate these changes in terms of the animal's ability to hear, the action of other agents can be more readily determined.

It is not impossible to examine microscopically a section through the cochlea and ascertain the degree to which the vessels of the stria vascularis are distended. Results obtained in this way are qualitative and frequently misleading. However, since various drugs have been thought to produce vascular stasis within the cochlea, the problem seemed worthy of a quantitative study. A number of points arise when an analysis of this type is attempted. What is the proportion of vascular to total stroma in the stria vascularis for control animals, and is it different for the various whorls of the cochlea? Does one drug produce a relative vascularity of greater significance than another drug, and, if so, is the change equally distributed throughout the cochlea? Does a distention of the vessels in the stria vascularis alter the pressure of the intracochlear fluid and so influence hearing?

The present study is an attempt to estimate in relative terms the vascularity of the cochlea for control animals and the influence which drugs play in altering this determination.

Supported by a grant from the John C. and Edward Coleman Memorial Fund, University of California.

From the laboratories of the George Williams Hooper Foundation for Medical Research, University of California.

1. Taylor, H. M.: The Neural Mechanism of Hearing: "Nerve Deafness" of Known Pathology and Etiology; Deafness from Drugs and Chemical Poisons. Laryngoscope **47**:692-705, 1937.

METHOD

The cochlea of the guinea pig has been selected for this study because of the numerous whorls, which offer an opportunity to compare the relative vascularity of the stria vascularis from base to apex. Furthermore, my previous studies on drugs have made available for this investigation a considerable number of sections of the cochlea of this animal. The control group of animals and the animals which received drugs ranged in weight from 250 to 410 Gm. They were perfused with Regaud solution under a constant pressure, as previously described (Covell [2]). Serial sections of the pyroxylin-embedded cochlea of each animal were stained either with a modified Van Gieson stain or with Harris' hematoxylin.

The three guinea pigs which received quinine were given daily subcutaneous injections of the drug (200 mg. per kilogram of body weight) in a 2.5 per cent solution for fifty-seven days. The three animals which received sodium salicylate (same dosage and concentration as quinine) were given daily subcutaneous injections for fifty-three days. The animals given injections of codeine phosphate received the drug for thirty-six days (15 mg. per kilogram of body weight) in a 0.3 per cent solution. Soluble barbital U. S. P. (sodium barbital) in a 2.5 per cent solution and a dose of 200 mg. per kilogram of body weight was administered daily for thirty-seven days to three guinea pigs.

Estimation of Relative Vascularity.—The stria vascularis of every fifth section (10 microns) was outlined by means of a camera lucida at a magnification of 400 diameters. Only midvertical sections of the cochleae were selected for study. Tangential sections through the stria vascularis were not included in the estimations. The appearance of the organ of Corti served as a guide for judging whether or not a section was tangential. The outlines of the stria vascularis and the vascular areas were made for a single optical plane on standard weight paper of constant thickness. The stratum fibrosum striae with its vessels was not included. The outlines of the stria vascularis, including those of the vascular areas, were cut out and weighed on an analytic balance. The total weight, which was determined for each turn of the cochlea, corresponded to the combined weight of the protoplasm of the stria vascularis and of the vascular areas. Since the amount of the stria vascularis in the most apical part of the guinea pig's cochlea is variable, estimates of it were included with the third turn. To determine the actual surface area represented for the different whorls, the weight of the paper per square centimeter was divided into the weight of the paper for each turn and the result further divided by the magnification squared. Each approximation represented about twelve sections through the cochlea.

For checking the accuracy of this procedure every section (midvertical) of two cochleae was outlined. It was found that the results obtained agreed within 0.6 per cent with the estimations of relative vascularity in the stria vascularis for every fifth section. This variation was not considered of enough significance to justify the effort involved in utilizing every section.

Sources of error to be considered in the use of such a method are: shrinkage of tissue due to fixation, individual variation in the use of a camera lucida, variations in opinion as to what might constitute a midvertical section through the cochlea and variations in the weight of a paper under different atmospheric conditions.

2. (*a*) Covell, W. P.: A Cytologic Study of the Effects of Drugs on the Cochlea, Arch. Otolaryng. **23**:633-641 (June) 1936. (*b*) Covell, W. P., and Noble, L.: The Significance of Myelin Sheath Degeneration for the Cochlear Nerve, Ann. Otol., Rhin. & Laryng. **46**:896-911, 1937.

Precautions were taken to eliminate as many of these sources of error as possible. The results of any attempt to determine the vascularity of a tissue or organ in quantitative terms are bound to show considerable variability. At best the value obtained is only an approximation.

RESULTS

Determinations of the vascular surface areas in percentage of the total surface areas of the stria vascularis for midvertical sections through the cochleae of control and drug-treated animals are given in the table. The basal whorls for many of the cochleae of the control animals reveal a slightly increased value over the estimates for the other turns of the same cochlea. This is not considered significant, and the relative value

Number of Animal	Drug	Vascular Surface Area × 100 / Total Surface Area of Stria Vascularis			Deviation from Weighted Mean Value (5.95%) for Controls	Ratio of Vascular Surface Areas to Protoplasmic Surface Areas	Deviation (Percentage) from Mean Ratio Value (1:16.45) for Controls
		Base	Middle	Apex			
LB1	.....	6.0	5.0	5.8	0.15	1 : 15.88	3.46
LB2	.....	5.1	5.3	5.6	0.62	1 : 17.78	8.08
LB3	.....	6.6	6.4	5.7	0.28	1 : 15.10	8.21
LB4	.....	6.5	6.3	6.0	0.32	1 : 14.97	8.88
LB5	.....	6.1	5.3	5.4	0.35	1 : 16.93	2.91
LB6	.....	5.5	5.2	5.1	0.68	1 : 18.01	9.48
LQ1	Quinine	15.1	13.1	14.1	8.15	1 : 6.11	62.86
LQ2	Quinine	9.1	8.5	11.7	3.82	1 : 9.51	42.19
LQ3	Quinine	12.7	14.4	13.9	7.72	1 : 6.33	61.52
LS1	Sodium salicylate	12.4	11.6	13.5	6.55	1 : 7.03	57.26
LS2	Sodium salicylate	14.2	13.6	12.7	7.55	1 : 6.42	60.97
LS3	Sodium salicylate	10.8	9.3	10.2	4.15	1 : 8.94	45.65
LC1	Codeine	8.3	6.6	7.1	1.38	1 : 12.76	21.82
LC2	Codeine	10.4	7.1	8.3	2.65	1 : 10.92	33.62
LC3	Codeine	9.5	9.9	8.7	3.42	1 : 9.71	41.28
LN1	Soluble barbital	5.9	5.0	4.0	0.85	1 : 18.89	14.83
LN2	Soluble barbital	5.2	4.7	4.9	1.02	1 : 19.31	17.31
LN3	Soluble barbital	4.4	4.8	4.7	1.32	1 : 20.61	25.29

determined as the weighted mean of all turns for the six animals is 5.95 per cent. The percentage values range from 5 to 6.6. The ratio of vascular surface areas to protoplasmic surface areas for all turns ranges from 1 : 14.97 to 1 : 18.01, and the mean value is 1 : 16.45.

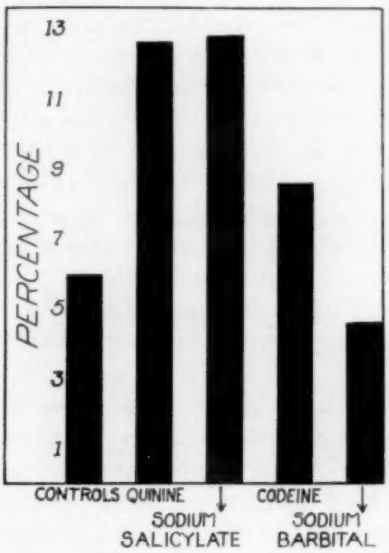
Similar determinations for animals which had received quinine for several weeks revealed significant changes in the values obtained for the relative vascularity. For two of these the percentages were from two to two and a half times greater than estimates for control animals. In one instance only was the figure approximately one and a half times greater. One might readily expect the ratio of vascular to protoplasmic surface areas to be decidedly altered. These estimates ranged from 1 : 6.11 to 1 : 9.51.

The relative values obtained for the animals treated with sodium salicylate were of the same order of magnitude as those for the group

of three animals treated with quinine. The percentage of vascularity for all turns of the cochleae ranged from 9.3 to 14.2.

The results for animals given injections of codeine showed less increase than those for animals treated with the drugs previously mentioned. Two of the estimations showed slightly more vascular surface area in the basal turns of the cochleae than in the middle and the apical turns. The analysis for a third animal revealed less variation for each turn. The ratios of vascular to protoplasmic surface area for the three animals ranged from 1:9.71 to 1:12.76.

Estimations of the relative vascularity for the cochleae of the guinea pigs which had been given injections of soluble barbital ranged from



Graphic representation of the relative vascularity of the stria vascularis for control and for drug treated animals. The first column represents control animals, and the others, animals treated with quinine bisulfate, with sodium salicylate with codeine and with soluble barbital, respectively.

4 to 5.9 per cent. These values were less than the range of 5 to 6.6 per cent obtained for the control group. Similarly, the ratios for the protoplasmic surface areas were approximately 15 to 25 per cent greater than the estimate of 1:16.45 for control animals.

A graphic representation of the relative vascularity of the stria vascularis for control and for drug-treated animals is shown in the figure. Approximately 6 per cent of the total surface area of the stria vascularis was vascular in the animals which did not receive drugs. This value was doubled after the administration of quinine and of sodium salicylate. Codeine increased the value less than half. Soluble barbital apparently

did not alter the relative vascularity in the same direction. There appeared to be less vascular surface area in the stria vascularis for each animal on which determinations were made. It is probable that this drug may cause a constriction of the arterioles in the stria vascularis. The difference of approximately 1 per cent between the estimations for this group and for the control group does not seem to be large enough to bear much significance.

COMMENT

It has previously been shown (Covell [2a]) that quinine bisulfate and sodium salicylate produce degenerative changes in the cytologic constituents of the cells of the stria vascularis. Mitochondrial preparations of the cochleae of a group of animals which had received various other drugs failed to reveal the same changes. Included in the group were animals which had been given injections of codeine and of soluble barbital. Since the study here reported has shown codeine to produce a distention of the vessels within the stria vascularis, one might expect cytologic evidence of its action to be apparent. It is probable that many different kinds of agents produce a vascular stasis for which microscopic studies fail to reveal further significant changes. It is also to be considered that any lack of evidence of a pathologic lesion in an experimental animal does not mean that the same agent could not produce an impairment of hearing in man. This might apply to a person who exhibits idiosyncrasy to a certain drug or to one in whom a loss in hearing is already established.

Vascular stasis within the cochlea would theoretically result in lack of proper nourishment to cochlear structures. It might alter the pressure of the intracochlear fluid, unless intracranial connections through the aquaeductus cochleae or displacement of the membrane of the round window could compensate for the increased pressure. Kobrak [3] found that contractions of the muscles of the middle ear resulted in displacement of the labyrinthine fluid. He found that nicotine produced a tetanic contraction of the tensor tympani muscle and caused a strong movement of the labyrinthine fluid. It is equally possible that many changes in the pressure of the fluid arise from sources within the cochlea.

The recent investigations of Belemer [4] on the distribution of small vessels within the stria vascularis have shown that, in addition to veins and capillaries, arterial vessels pass through its substance and emerge as arterioles. He expressed the belief that a mechanism of accommodation may exist for which movements of the basilar membrane cause

3. Kobrak, H.: Influence of the Middle Ear on Labyrinthine Pressure, Arch. Otolaryng. **21**:547-560 (May) 1935.

4. Belemer, J. J.: The Vessels of the Stria Vascularis with Special Reference to Their Function, Arch. Otolaryng. **23**:93-97 (Jan.) 1936.

a constriction of the arterioles as they emerge from the stria vascularis. In his opinion the resulting vascular distention within the stria vascularis may alter the pressure of the intracochlear fluid. On the basis of this hypothesis, it is possible to assume that agents which temporarily or permanently produce a vascular stasis may, at the same time, upset such a mechanism of accommodation. Drugs which act on the vascular endothelium or the vasomotor mechanism to cause a distention of the vessels could result in changes in pressure of the cochlear fluid.

These assumptions do not explain the complete action of drugs such as quinine and sodium salicylate. Evidence seems to show additional actions, as direct protoplasmic poisons or, in the case of quinine, indirectly through an intermediate metabolite (Findlay [5]). Substances which reach the inner ear through the blood stream first produce cytologic evidence of their reactions in the more basal turns of the cochlea. It is obvious from examination of a section through the cochlea that the lower half contains the greater volume of stria vascularis, an estimate of which awaits further study by quantitative methods. It may finally be possible to associate a loss in hearing with changes in vascularity.

5. Findlay, G. M.: Recent Advances in Chemotherapy, Philadelphia, P. Blakiston's Son & Co., 1930, p. 138.

DIFFERENTIAL DIAGNOSIS OF BACTERIAL MENINGITIS OF AURAL AND OF NASAL ORIGIN

JOSEPH C. YASKIN, M.D.
PHILADELPHIA

Bacterial meningitis occurs frequently and is one of the most important acute infections of the central nervous system. While it usually occurs independently of any primary disease of the ear and the nose, statistical study seems to indicate that, at least in general hospitals, the occurrence of meningitis as a complication of an otorhinologic condition is unpleasantly frequent as compared with its occurrence from other causes. The following tabulation, taken from Williams and Gasling,[1] shows that the meningococcus and the tubercle bacillus are the most common causes of meningitis in the metropolitan area of the city of New York. The frequency of meningococcic meningitis is undoubtedly augmented by epidemics, while tuberculous meningitis is prevalent in the poorer sections of congested urban districts.

Cause of Meningitis (Cases Studied Between 1910 and 1930 in the City of New York) (Williams and Gasling [1])

Meningococcus	1,151	Anaerobic bacillus	6
Tubercle bacillus	911	Sporotrichum	1
Pneumococcus	191	Torula	2
Streptococcus	164	Bacillus pyocyaneus	4
Influenza bacillus	90	Micrococcus catarrhalis	3
Staphylococcus	23	Diphtheroid bacillus	1
Bacillus coli	6	Mixed infection	17
Friedländer's bacillus	4	Cause not known	99
Typhoid bacillus	1		
Total			2,674

A statistical study of 123 cases of bacterial meningitis occurring in Philadelphia between 1920 and 1937, 96 of which were in the Graduate Hospital of the University of Pennsylvania, shows an entirely different picture (table 1). It will be noted in table 1 that 123 cases of meningitis from all causes are presented. In 54 of these cases the condition was not a complication of any frank infection of the ear and the nose; in 37 it was a complication of disease of the temporal bone; in 21 a com-

From the Department of Neurology, Graduate School of Medicine, University of Pennsylvania.

Presented as a part of a symposium on bacterial meningitis before the Section of Otolaryngology of the College of Physicians of Philadelphia, Nov. 17, 1937.

1. Williams, A. W., and Gasling, R.: Bacteriology of Meningitis, A. Research Nerv. & Ment. Dis., Proc. **12**:353, 1932.

plication of disease of the paranasal sinuses, and in 11 a sequela of cranial trauma. This analysis shows that in a large number of cases in general hospital practice bacterial meningitis is a complication of an infection of the ear or nose and that the predominant organisms are the streptococcus, the pneumococcus and the staphylococcus.

The diagnosis of bacterial meningitis depends on the existence of the meningeal syndrome, on laboratory procedures and mainly on the demonstration of the organism in the spinal fluid. Clinicians and especially otorhinologists are interested in an early diagnosis and are frequently confronted with doubtful data. The existence of disease of the ear or the nose does not necessarily indicate that this condition is the cause of coexisting bacterial meningitis. However, as was pointed out by Dennet,[2] it may be the complication of primary meningitis. This is especially true in infants. One must bear in mind the common occur-

TABLE 1.—*Analysis of Cases of Bacterial Meningitis from 1920 to 1937* *

Organism	Origin				
	Systemic or Unknown	Temporal Bone	Paranasal Sinuses	Cranial Trauma	Total
Meningococcus	10	..	..	..	10
Tubercle bacillus	21	..	..	..	21
Pneumococcus	6	11	4	3	24
Streptococcus	6	16	7	3	32
Influenza bacillus	4	..	..	..	4
Staphylococcus	3	4	5	3	15
Friedländer's bacillus	1	..	..	..	1
Unknown	3	6	5	2	16
Total	54	37	21	11	123

* Of the 123 cases, 96 were from the Graduate Hospital of the University of Pennsylvania, while the remaining 27 were from other hospitals and from private practice.

rence of chronic aural disease and especially of sinus disease and evaluate various neurologic conditions occurring in these subjects. There are a number of meningeal syndromes not due to bacterial meningitis which are associated with disease of the ear or the nose but are entirely independent of it. These include spontaneous subarachnoid hemorrhage, acute syphilitic meningitis and virus infections. On the other hand, there are complications of infections of the ear or the nose which give rise to meningeal syndromes but which are not of bacterial origin. These and other sources of error in diagnosis will be discussed. The clinical features of meningeal involvements will be considered first.

THE SYNDROME OF MENINGEAL IRRITATION

Meningeal irritation may be produced by a great variety of causes, such as exogenous poisoning, systemic toxemia, virus and bacterial

2. Dennet, R. H.: Meningitis in Infancy, A. Research Nerv. & Ment. Dis., Proc. **12**:481, 1932.

intoxication and infection, epidural infection in the brain and the spinal cord and certain types of tumor of the brain. The common symptoms (headache, dizziness and vomiting) and focal signs of meningeal irritation (rigidity of the neck and Kernig's and Brudzinski's sign) are well known but require careful clinical evaluation. Thus, rigidity of the neck is often observed in normal infants. I have observed rigidity of the neck in 2 cases of early cervical adenitis in which the diagnosis of meningitis was seriously considered. Recently a "Kernig sign" was found to be due to scurvy. In older persons arthritis of the spine may give rise to pseudomeningeal signs. On the other hand, I have observed 2 cases of purulent meningitis (both streptococcic) and at least 3 cases of tuberculous meningitis in which at the height of the disease there was no rigidity of the neck and no Kernig or Brudzinski sign. In cases in which the diagnosis is doubtful, therefore, a lumbar puncture is indispensable for accurate diagnosis. It is generally known that meningeal signs may be absent early in the course of meningitis and that they often disappear in the terminal stages.

In the final analysis, it is the examination of the spinal fluid which determines the type and the cause of meningeal irritation. There are many basic causes for the various changes in the spinal fluid. Of these, three are most important: the changes in the blood plasma, the changes in the hemato-encephalic barrier and the invasion of the ventriculo-subarachnoid cavities by foreign elements.

The changes in the spinal fluid are summarized in table 2.

Acute Meningeal Symptoms Due to Agents Other than Known Bacteria.—There are a number of conditions associated with evidence of acute meningeal irritation and not due to bacterial invasions:

1. Cervical adenitis and retropharyngeal abscess have in my experience been misdiagnosed as acute meningitis. The chief source of error in connection with these conditions is the limitation of movements of the neck. One rarely sees rigidity of the neck and the back in association with a tumor of the basal and the posterior fossa of the brain or with a spinal epidural abscess or tumor. Infiltration of the meninges by lymphoblastomas (Hodgkin's disease, leukemia) rarely gives rise to meningeal irritation.

2. Meningismus (acute serous meningitis) occurs in the course of acute systemic infections, such as pneumonia, measles, influenza, gastroenteritis, acidosis, tetany and bacterial endocarditis, and is characterized by elevation of the pressure of the spinal fluid and occasionally by an increase in the protein content, but the cell count remains normal and organisms are absent. Meningismus is seen in association with toxic conditions, such as delirium tremens, uremia and congestive cardiac disease. Meningismus is always observed when air is injected into the

TABLE 2.—*Usual Changes in the Cerebrospinal Fluid, Associated with the More Common Syndromes of Acute Meningeal Irritation*

Meningeal Syndrome	Pressure	Character	Cells, per Cu. Mm.	Protein, Mg. per 100 Cc.	Reducing Substances Reported as Sugar, Mg. per 100 Cc.	Chlorides, Mg. per 100 Cc.	Bacteria	Colloidal Gold Curve	Wassermann Reaction
None	75 to 150 H_2O	Clear; colorless	0 to 10 lymphocytes	15 to 40	40 to 60	720 to 750	None	Normal	Negative
Acute serous meningitis	Increased	Normal	Normal or slight increase	Normal or slight increase	Normal	Normal	None	Normal	Negative
Virus diseases; acute anterior poliomyelitis	Increased	Normal	0 to 1,000; first 24 hours polymorphonuclears later lymphocytes	Increase in majority of cases	40 to 120	Normal	None	Normal or in zone 2	Negative
Virus diseases; epidemic encephalitis; disseminated encephalomyelitis	Normal or increased	Normal	Normal or slight increase in lymphocytes	Slightly increased	40 to 120	Normal	None	Normal or in zone 2	Negative
Virus diseases; benign lymphocytic choriomeningitis	Moderately increased	Clear	50 to several thousand lymphocytes	Increased	Normal or slightly increased	Normal	None	Normal or in zone 2	Negative
Spontaneous subarachnoid hemorrhage	Markedly increased	Bloody or xanthochromic	Many red cells	Markedly increased	Normal or increased	Normal or increased	None	In zone 2 to 4	Negative
Acute syphilitic meningitis	Normal to 700	Clear	25 to 2,000 lymphocytes; polymorphonuclears predominant in 5 to 17 per cent of cases	Increased	18 to 84	636 to 742	None	In any of the four zones	Positive in 90%
Aseptic meningitis	Usually increased	Usually clear	25 to several thousand, usually polymorphonuclear but rarely may be lymphocytes	Moderately or considerably increased	Normal	Normal	None	Normal or in meningitic zone	Negative
Tuberculous meningitis	Usually increased	Normal or opalescent	80 to 1,000 lymphocytes; rare polymorphonuclears	100 to 1,000	0 to 40, usually decreased	Decreased in the majority of cases	Found in over 50% of cases	In meningitic zone	Negative
Purulent meningococcic, pneumococcic, staphylococcic or streptococcic meningitis	Markedly increased	Slightly cloudy to thick pus	50 to several thousand polymorphonuclears	Markedly increased	0 to 40, usually decreased	Normal or slightly increased	Present	In meningitic zone	Negative

subarachnoid space and occasionally follows spinal anesthesia. In some infections, such as typhoid fever, the meningeal signs are accompanied with pleocytosis, and there is actually acute meningo-encephalitis.

3. Otitic hydrocephalus is a rare form of lasting serous meningitis characterized by persistent elevation of the pressure of the spinal fluid, elevation of the optic disks, headache and vomiting. In one case which came under my observation this disease followed mastoiditis and lateral thrombophlebitis. The child made an uneventful recovery in several months.

4. Virus diseases (poliomyelitis, epidemic encephalitis, disseminated encephalomyelitis and benign lymphocytic choriomeningitis) are associated with varying degrees of meningeal irritation. In cases of poliomyelitis there is in the first forty-eight hours a definite increase in cells. In cases of epidemic encephalitis and of disseminated encephalomyelitis there is usually no pleocytosis. Benign lymphocytic choriomeningitis [3] ooccurs in epidemic form and is characterized by an acute onset of meningeal signs, a marked increase of lymphocytes in the spinal fluid with absence of bacteria, a short, mild course without any complications and termination usually in recovery in from seven to ten days.

5. In hemorrhagic conditions free blood in the subarachnoid space gives rise to acute meningeal symptoms. In 2 cases I have observed spontaneous subarachnoid hemorrhage in association with acute sinusitis and clinically closely resembling acute bacterial meningitis. Spontaneous subarachnoid hemorrhage is generally due to congenital aneurysm and in recent years has been common. Subarachnoid bleeding is observed commonly in association with cranial trauma, or with intracerebral hemorrhage and less commonly with purpura, hemophilia, drug and metallic poisoning or a severe systemic infection.

6. Acute meningeal involvement in the course of syphilis is common, but it is usually patchy and chronic and is not accompanied with any evidences of acute meningitis. Evidences of acute meningitis in cases of secondary syphilis are not uncommon, while the disease occurs in 5 per cent of cases of latent syphilis.[4] In cases of acute syphilitic meningitis are found rigidity of the neck, the Kernig sign, an increase of cells—predominantly of lymphocytes, changes in the colloidal gold curve and, in 90 per cent, a positive Wassermann reaction. In some cases there may be a considerable rise in temperature. The history, the associated neurologic findings, especially the pupillary changes in cases in which the involvement is latent, and the results of examination of the spinal fluid and of the blood establish the diagnosis.

---

3. Dummer, C. M.; Lyon, R. A., and Stevenson, F. E.: Benign Lymphocytic Meningitis (Aseptic Meningitis), J. A. M. A. **108**:633 (Feb. 20) 1937.

4. Merritt, H. H., and Moore, M.: Acute Syphilitic Meningitis, Medicine **14**: 1, 1935.

7. Aseptic (sympathetic, protective or reactive) meningitis is probably the type of the greatest importance to otorhinologists. Clinically, it is manifested by rigidity of the neck, the Kernig sign, some elevation in temperature, an increase in the pressure of the spinal fluid, which is clear or opalescent, an increase of cells (usually of the polymorphonuclear type and rarely lymphocytes), an increase in total proteins, no appreciable change in the sugar or the chloride content and absence of organisms. This condition is occasionally observed in the absence of any disease of the ear or the accessory sinuses. When such disease is present, the condition may be associated with epidural abscess, circumscribed meningitis, intracerebral or cerebellar abscess or sinus thrombosis, or it may be the beginning of bacterial meningitis before the bacteria are detectable by present laboratory procedures. I have observed an exceedingly large number of cases in which the condition was first diagnosed as aseptic meningitis, but my records reveal only 9 cases in which recovery followed without the finding of bacteria in the spinal fluid or the development of other intracranial complications. Of these 9 cases, in 7 the disease was associated with mastoiditis; in 2 it preceded mastoidectomy; in 5 it followed mastoidectomy, and in 4 it required revision of the operative field. In the remaining 2 cases the condition was associated with infection of multiple sinuses, and recovery followed without any major surgical procedures. In the vast majority of cases in which a diagnosis of aseptic meningitis was made at the beginning, the condition was proved by subsequent studies of the spinal fluid to be bacterial meningitis, while in some there was abscess of the brain, epidural abscess or sinus thrombosis.

The early diagnosis of aseptic meningitis is obviously desirable for therapeutic reasons. The absence of organisms is the chief differential point. However, a decrease in the sugar content and, according to Kopetzky and Fishberg,[5] an increase in potassium salts, a lowered p_H, a lowered carbonate content and particularly a marked increase in the lactic acid content of the spinal fluid favor the diagnosis of bacterial meningitis. Kopetzky [6] stated that these chemical changes may indicate invasion but no actual tissue reaction (meningitis) and that after complete eradication of the foci of origin, recovery may follow without the development of meningitis.

Bacterial Meningitis.—In the majority of cases bacterial meningitis complicating disease of the ear or nose constitutes an empyema of the ventriculosubarachnoid system, which affords no difficulty in diagnosis. The history of the primary infection, the systemic reactions (fever, prostration and leukocytosis), the evidences of meningeal irritation and

5. Kopetzky, S. J., and Fishberg, E.: Early Diagnosis in Meningitis: Chemical Studies of Spinal Fluid, Arch. Otolaryng. **16**:851 (Dec.) 1932.

6. Kopetzky, S. J.: The Clinical Picture of Bacterial Meningitis with Particular Reference to Its Changing Phases, Laryngoscope **47**:301, 1937.

the spinal fluid findings of purulent meningitis (table 2) are definitely clearcut. Since purulent meningitis means pathologically meningoencephalomyelitis, the patient has, in addition to headache, mental symptoms, various motor and sensory signs, incontinence of urine, etc.

Of the 58 cases in our series in which the condition was due to disease of the ear or nose (table 1) the organism found was a streptococcus in 23, a pneumococcus in 15 and a staphylococcus in 9. In 11 either the organism was not specifically identified or it was not found, but the clinical course was that of severe meningitis and the termination was fatal. Without the aid of studies of the spinal fluid it is impossible to differentiate the various types of purulent meningitis. While streptococci, pneumococci and staphylococci are the most commonly observed organisms, there are many others which invade the ventriculosubarachnoid system through the pathways of the nose and ear.

Bacterial meningitis of aural and of nasal origin should be differentiated from the acute meningeal syndromes resulting from (*a*) nonbacterial causes which already have been discussed and (*b*) bacterial infections not of aural or nasal origin, especially meningococcic and tuberculous meningitis. Both of these types are common and may occur in persons with aural or nasal disease.

Meningococcic meningitis occurs in both epidemic and sporadic form. When occurring in epidemic form a considerable proportion of septicemic types may be observed, as was the case in the Detroit epidemic.[7] In infants it may be masked by coexisting aural disease.[2] The association of cerebrospinal fever with severe nasopharyngitis has been recognized for many years.

The clinical course of meningococcic meningitis varies to a marked extent. It may be so mild as to allow the patient to remain ambulatory or so severe as to terminate fatally in from twenty-four to forty-eight hours. In addition to neurologic signs indicative of meningitis and meningo-encephalitis, purpuric spots, herpes and the swelling of joints are frequently observed. The finding of the specific organism in the spinal fluid establishes the etiologic diagnosis.

From the standpoint of treatment, meningococcic meningitis should be regarded as an emergency, for the earlier the diagnosis is made and the sooner the treatment is started, the better are the chances for recovery. In the great majority of cases the specific organism can be identified on direct smear and culture. When doubt exists, the condition should be treated as meningococcic meningitis.

Tuberculous meningitis usually occurs in children and represents a terminal process of general miliary tuberculosis. Less commonly it is secondary to foci in the lymph nodes, the bones, the lungs and other structures. The lesions are located mainly in the sylvian fissure and

7. Gordon, J. E.: A. Research Nerv. & Ment. Dis., Proc. **12**:453, 1932.

others, under the cerebellum and at the base of the brain. Tuberculous meningitis is usually nonpurulent.

The onset may be sudden but is usually insidious and is masked by gastro-intestinal symptoms, especially causeless vomiting, and by change in disposition, especially irritability and drowsiness. Then follow irregular fever, involvement of the cranial nerves, the syndrome of meningeal irritation and varied evidences of meningo-encephalitis. Choroid tubercles are rarely encountered. The spinal fluid is under considerable tension. It is usually clear but forms a "pellicle" when permitted to stand for several hours; the number of cells is markedly increased, and they are usually lymphocytes; the protein content is always increased; the sugar and the chloride content show progressive diminution, and the tubercle bacillus may be recovered in 50 per cent of cases. The Levinson test gives positive results in a large percentage of cases. The tryptophan test [8] is said to give positive results in most cases, even in the early stages. Inoculation of guinea pigs is often valuable.

I have observed a number of cases in which the manifestations were atypical. In some there was no meningeal syndrome; in others there was an increase in polymorphonuclears, and in a few a decrease of sugar or of chlorides in the spinal fluid was not observed. In all the cases in my experience tuberculous meningitis terminated fatally.

SUMMARY

Meningitis originating as a complication of nasal or aural disease in 58 of 123 cases in general hospital practice was found to be bacterial. The organisms isolated in these cases were streptococci, pneumococci and staphylococci. Statistics from large metropolitan districts indicate, however, that meningococcic and tuberculous meningitis are more common than any other form.

The diagnosis of bacterial meningitis of nasal or aural origin depends on the presence of the meningeal syndrome and changes in the spinal fluid. This form of meningitis should be differentiated from (*a*) conditions not due to invasion by known bacteria, such as cervical adenitis, retropharyngeal abscess, meningismus, virus diseases, hemorrhagic conditions, acute syphilitic meningitis and aseptic meningitis, and (*b*) other forms of bacterial meningitis, of which the commonest are meningococcic and tuberculous.

False signs, absence of signs and unusual manifestations in the spinal fluid are cited to emphasize the need for careful clinical evaluation in the individual case. The prevalence of aural and nasal disease in association with many etiologically unrelated meningeal syndromes is stressed as a further reason for appropriate clinical orientation.

8. Pongratz, R.: Importance of Tryptophan Test for Diagnosis of Tuberculous Meningitis, München. med. Wchnschr. **84**:814, 1937.

NASAL IONIZATION

HISTOLOGIC STUDIES IN RELATION TO CLINICAL EVALUATION

A. R. HOLLENDER, M.D.
AND
NOAH D. FABRICANT, M.D.
CHICAGO

Ionization is not a new procedure in rhinologic practice. Leduc [1] in 1908 suggested its use for intumescent rhinitis, and later Friel [2] advocated the treatment for certain conditions of the nose and the sinus. It is probable that other European and American workers early employed nasal ionization, without placing their results on record. In 1926 Cottle and one of us [3] published a report on the efficacy of zinc ionization in relieving the milder forms of hypertrophic rhinitis. Although the method had been tried in cases of vasomotor rhinitis, a lack of uniform results made it undesirable to present this phase of the problem to the medical profession. Since then there have been periods during which ionization has been enthusiastically recommended not only for nasal allergy but for a large variety of diseases in other parts of the body.[4]

Credit for the popularization of ionization in its application to nasal allergy belongs to Warwick.[5] While his technic differed somewhat from that which had been utilized in the treatment of nonallergic conditions,

---

Read before the Chicago Laryngological and Otological Society, Nov. 1, 1937.

From the Department of Laryngology, Rhinology, and Otology, University of Illinois College of Medicine.

EDITOR'S NOTE.—This paper was accepted for publication although its thesis is at variance with some generally accepted ideas in regard to the effect of ionization. It is published, therefore, as a matter of fairness to both sides in a controversy on histopathology.

1. Leduc, Stephane: Electric Ions and Their Use in Medicine, translated by R. W. MacKenna, London, Rebman, 1908.

2. Friel, A. R.: Treatment of Sepsis in Nose and Ear by Ionization, Practitioner **103**:449 (Dec.) 1919.

3. Hollender, A. R., and Cottle, M. H.: Recent Advances in the Treatment of Nasal Accessory Sinus Disease, Eye, Ear, Nose & Throat Monthly **5**:575 (March) 1926; Physical Therapy in Diseases of the Eye, Ear, Nose and Throat. New York, The Macmillan Company, 1926.

4. Dyson, J. Newton: The Practice of Ionization, London, Henry Kimpton, 1936.

5. Warwick, H. L.: Treatment of Hay Fever and Its Allied Conditions by Ionization: Preliminary Report, Laryngoscope **44**:173 (March) 1934.

fundamentally it was the same. Today those who have become adherents of the method employ the original technic. This requires as equipment a simple source of galvanic current, an electrolyte (usually a 1 or 2 per cent solution of sulfate) and electrodes to convey the current to the nasal mucosa. Strips of gauze or cotton saturated with the electrolyte insure proper contact with the nasal membranes, while a wet felt-covered metal dispersive electrode placed on the forearm closes the circuit.

There has been manifested in some quarters a tendency to reject ionization because histologic studies have allegedly demonstrated deleterious changes in the tissue which were assumed to be permanent or to require considerable time for resolution. Our present study was undertaken as an extension of one begun in 1935 by Gorin and one of us.[6]

SUMMARY OF THE RECENT LITERATURE ON THE HISTOLOGIC PICTURE CAUSED BY NASAL IONIZATION

The histologic picture of the mucosa of the nose and the sinuses preceding and following ionization has been studied in recent years in animals and in man. After investigating late changes in the mucosa of the frontal sinuses and noses of dogs, McMahon [7] stated that within seven weeks after ionization of the epithelium it returned to normal. Fibrosis of the subepithelial tissue was progressively more marked in animals killed at intervals of from eighteen hours to seven weeks following ionization.

The final results of ionization of the human nose observed by Dean [8] were: fibrosis with thinning of the tunica propria; hyperplasia of the epithelium with some metaplasia; a partial destruction of glandular function, and a decided increase in the goblet cells. Dean claimed that there was often a thickening of the walls of the vessels, especially of the capillaries. Some of the acini of the glands appeared to be obliterated by the cellular hyperplasia. In others the secretory cells were atrophied. The basement membrane underwent marked thickening and was more dense.

Smit [9] summarized the changes in the tissues of the nasal mucosa of dogs and guinea pigs after ionization. He attempted to correlate these findings with changes that might occur in the human mucosa removed for biopsy before and after treatment. This, however, Smit stated, was difficult, because of the pathologic state existing prior to iontophoresis. He pointed out that the changes in the tissues were coagulative necrosis

6. Hollender, A. R., and Gorin, M.: Influence of Ionization on Vasomotor Rhinitis: Clinical and Experimental Studies, Illinois M. J. **68**:492 (Dec.) 1935.

7. McMahon, Bernard J.: Late Changes in the Mucosa of the Frontal Sinuses and Nose of Dogs Following Ionization, Arch. Otolaryng. **22**:454 (Oct.) 1935.

8. Dean, L. W.: The Fundamentals of Allergic Rhinitis with Particular Reference to Ionization, Ann. Otol., Rhin. & Laryng. **45**:326 (June) 1936.

9. Smit, H. M.: Tissue Changes in Iontophoresis, Ann. Otol., Rhin. & Laryng. **45**:138 (March) 1936.

of the epithelium; early stages of inflammation with polymorphonuclear leukocytes, which were later replaced by round cells; atrophy and loss of glands; metaplastic changes in glands and ducts; formation of stratified squamous epithelium, which later resumed a normal ciliated columnar character (if a chronic inflammatory process is not present); fibrosis of the submucosa; dehydration of edematous tissue; fibrosis of the blood vessels, and in some cases cartilaginous necrosis.

Shields [10] reported on the microscopic examination of the nasal mucosa in a number of cases of allergy before and after ionization. He found that there was a general tendency for the columnar epithelium to be replaced by stratified epithelium but that there was no marked fibrosis.

Boling [11] showed that after ionization of the sheep's nose there was complete regeneration of the ciliated epithelium. Wenner [12] has made the same observation on cats.

HISTOLOGIC STUDIES

The photomicrographs shown in figures 1 to 11 of sections removed from the nose are representative of numerous specimens collected in this study.

FACTORS PERTAINING TO NORMAL AND PATHOLOGIC NASAL MUCOSA

A critique of the methods heretofore employed in evaluating histologic studies of the results of nasal ionization must of necessity encompass three cardinal points: (1) the determination, if at all feasible, of the constancy of normal nasal epithelium in the human nose; (2) the method of interpreting the results of biopsy obtained in conventional practice, and (3) the advisability of making deductions and conclusions without more accurate data.

It will not be amiss to describe briefly the histologic structure of nasal mucosa. Schaeffer [13] defined normal nasal epithelium as of pseudostratified columnar type. Latta and Schall [14] have stated that normal nasal epithelium may undergo a variety of changes in its adaptability to ever changing circumstances. Nasal epithelium may be not only of the simple

10. Shields, Clive: Hay Fever with Special Reference to Treatment by Intranasal Ionization, London, Oxford University Press, 1937.

11. Boling, L. R.: Regeneration of Nasal Mucosa, Arch. Otolaryng. **22**:689 (Dec.) 1935.

12. Wenner, W. R., cited by Dean and others.[10]

13. Schaeffer, J. P.: The Nose, Paranasal Sinuses, Naso-Lacrimal Passageways and the Olfactory Organ in Man, Philadelphia, P. Blakiston's Son & Co., 1920.

14. Latta, J., and Schall, R.: The Histology of the Epithelium of the Paranasal Sinuses Under Various Conditions, Ann. Otol., Rhin. & Laryng. **43**:945 (Dec.) 1934.

ciliated columnar type but of the stratified ciliated, the cuboidal, the stratified cuboidal, the squamous or the transitional type. Latta and Schall deemed it reasonable to assume that there must be a variation in the character of the epithelium, and perhaps in that of the underlying structures of the normal nose, according to the age of the subject, the frequency of acute infections and such factors as weather conditions, smoke, dust and chemical substances.

Hilding [15] in reporting morphologic studies of the epithelium following variations in ventilation, brought up the question whether there are several types of epithelium in the upper part of the respiratory tract. He noted that there are areas in the anterior portion of the nose which exhibit little if any ciliary activity. By microscopic examination he observed that the epithelium of the inactive areas assumed the form of the stratified squamous type, without cilia. Oppikofer [16] examined the epithelium of the turbinates of 200 subjects and found islands of squamous or transitional epithelium. In another series of experiments Hilding suggested that the different forms of epithelium are not fixed types but are more or less interchangeable according to circumstances.

A clinically normal nose may on microscopic examination reveal evidence of epithelial metaplasia. Is it therefore to be assumed that a nose is clinically abnormal because the microscope shows a deviation from normal in the epithelium? It is our contention that normal epithelium seldom remains "normal" in the classic sense, since epithelium, exposed to continuous bombardment by multiple factors and conditions, is subject to change. There is, we believe, an urgent necessity for reexamining and reevaluating the concept of "normal" epithelium.

The thickness of the basement membrane in the normal human nose varies greatly. For the most part it is definitely well developed and well marked. In some instances it is so delicate as to appear absent, while, conversely, in chronic infections it may be markedly thickened. The question arises: What is the normal thickness of basement membrane? It is obvious that tissue taken for biopsy from one portion or area of the nasal mucosa may reveal a thinning or thickening different from that shown by tissue removed from an adjacent site.

The tunica propria, or stroma, consists of a loose fibro-elastic network of connective tissue, which under what are regarded as normal conditions shows a great variation in the amount of elastic fibers, ranging from a dense to a sparse formation.

---

15. Hilding, A.: Experimental Surgery of Nose and Sinuses: Changes in Morphology of Epithelium Following Variation in Ventilation, Arch. Otolaryng. **16**:9 (July) 1932.

16. Oppikofer, E.: Beiträge zur normalen und pathologischen Anatomie der Nase und ihrer Nebenhöhlen, Ztschr. f. laryng., Rhin. **19**:28, 1927.

Both the middle and the inferior turbinate are favorable sites for the taking of biopsy specimens. The mucosa of the middle turbinate varies in thickness, a low cuboidal or squamous variety of epithelium frequently replacing the ciliated epithelium, especially over the anterior tip. As in the case of the middle turbinate, the epithelium of the anterior

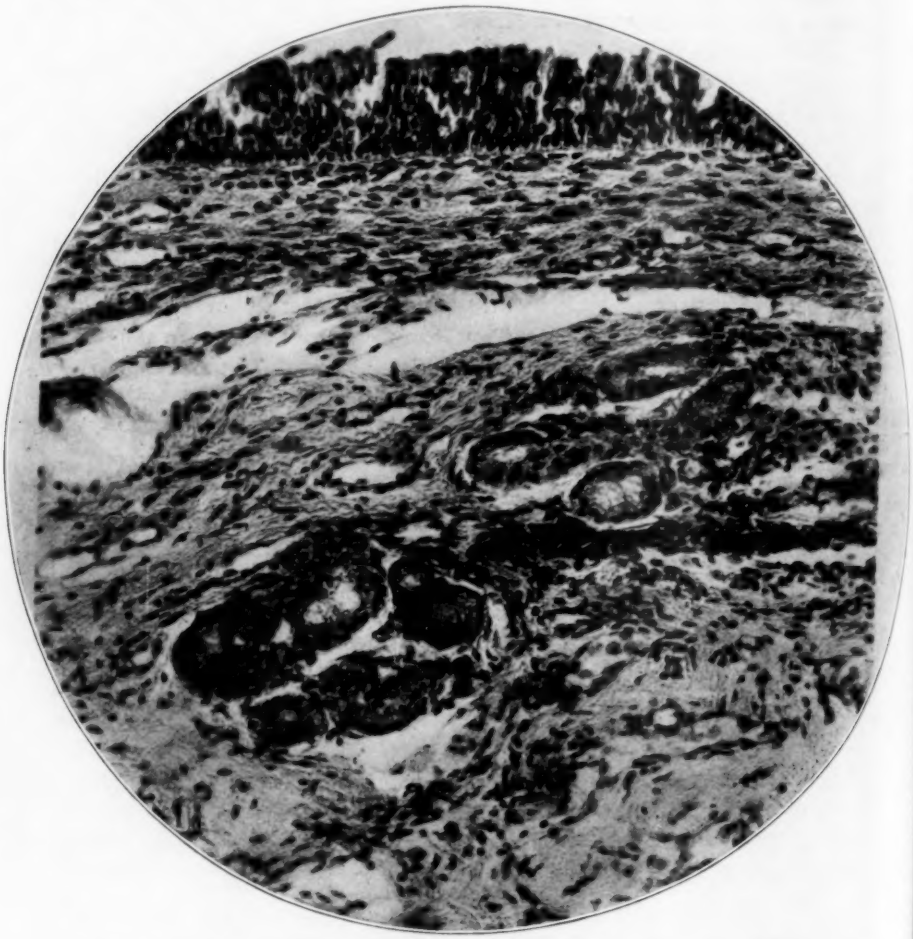


Fig. 1 (case 1).—Specimen of nasal mucosa removed in a case of vasomotor rhinitis before ionization. The lining epithelium is for the most part intact. Cilia are present in some areas and absent in others. The stroma is slightly infiltrated by small and large lymphocytes and a few plasma cells, while the mucus-secreting glands appear to be normal.

tip and the inferior border of the inferior turbinate may be replaced by a low cuboidal or squamous type of epithelium. Glands are numerous but not as numerous as in the case of the middle turbinate.

HISTOLOGIC PICTURE OF CHRONIC NASAL CONDITIONS

For the present we shall disregard the variety of mucosal changes seen in the clinically normal nose and consider the histologic picture present in certain chronic nasal conditions. The individual layers of

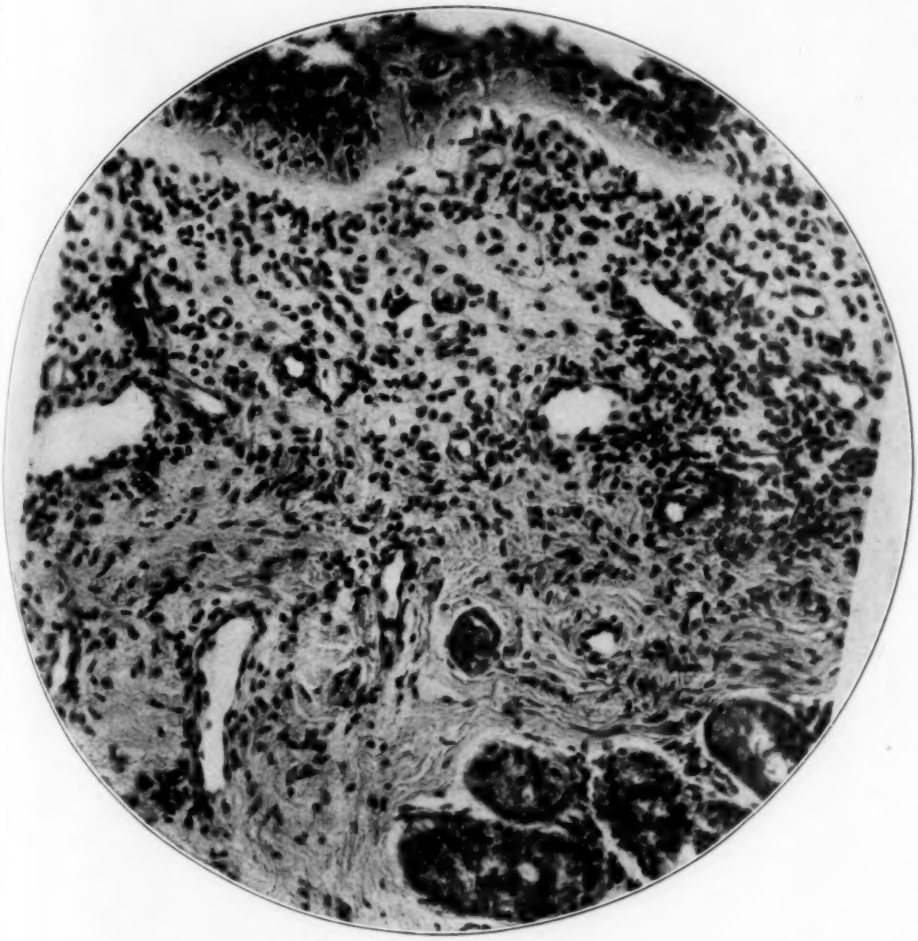


Fig. 2 (case 1).—Specimen of nasal mucosa removed in a case of vasomotor rhinitis immediately after ionization. The epithelium reveals swollen epithelial cells the nuclei of which stain more deeply than normal. There is complete disappearance of cilia. The basement membrane is markedly thickened. The round cell infiltration is increased. The mucus-secreting glands appear to be normal. (There was a forty-five minute interval between the taking of the specimen immediately preceding and immediately following ionization. It is obvious that the basement membrane could not be thickened so markedly in so short a period.)

mucosa show characteristic microscopic changes according to the severity and the duration of the infection. The epithelium undergoes a definite thickening, as does the basement membrane. The thickening, however, is not evenly distributed throughout the entire length of the nasal mucosa. The alterations in the subepithelial layer are governed mainly by the

Fig. 3 (case 2).—First biopsy specimen of nasal mucosa taken before ionization. The epithelium is of the ciliated columnar type. The stroma shows a round cell infiltration and an occasional eosinophil. The greatest part of the stroma is composed of fibrous connective tissue, which is moderately cellular and contains dilated lymph channels and transections of mucus-secreting glands.

density of the cellular infiltration. This layer is usually compact and firm, as a result of increased fibrous formation. Because of this, the glands frequently become compressed and undergo atrophic changes.

The walls of the larger blood vessels show a tendency toward fibrosis and thickening. Occasionally there is an increase in the capillary network.

The histologic changes which occur in cases of allergy are characterized by (1) edema which involves all the mucosal layers and (2) an

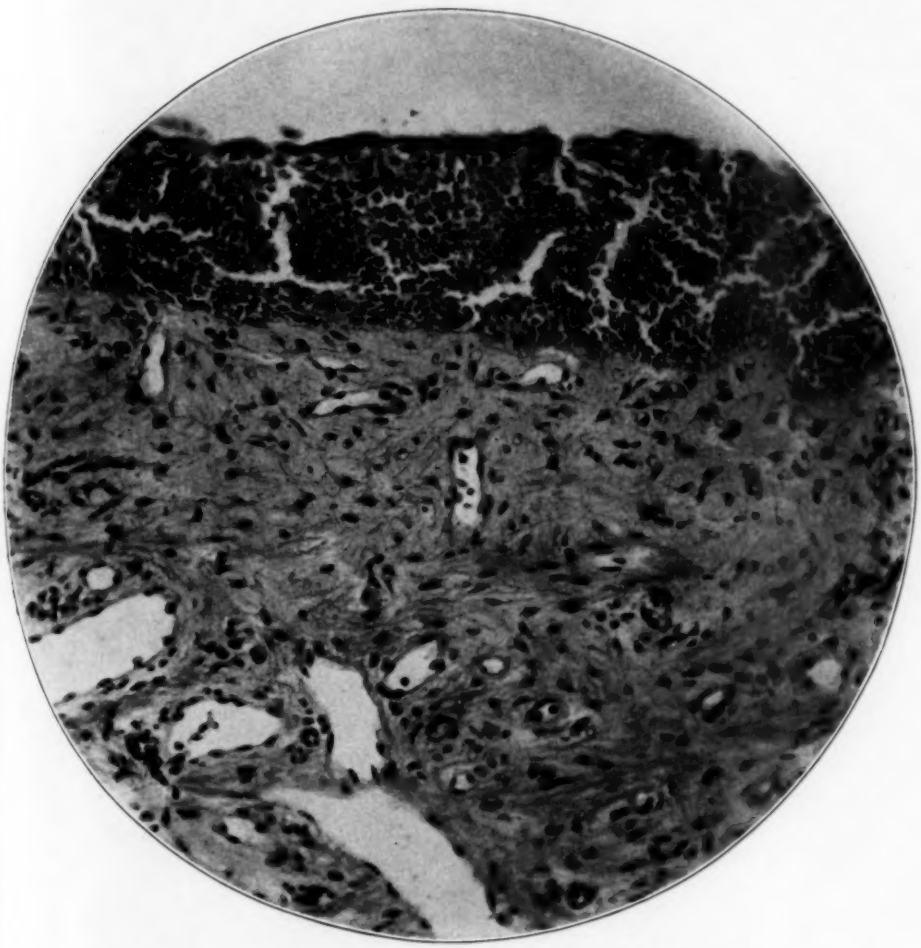


Fig. 4 (case 2).—Second biopsy specimen of nasal mucosa taken before ionization. The epithelium reveals definite metaplastic changes suggestive of the stratified squamous type. The underlying stroma is slightly edematous and shows a slight round cell infiltration. Dilated lymph channels may be seen. (Significant structural differences are evident in the epithelium and the underlying stroma in the first and the second specimen taken from the same subject before ionization.)

eosinophilic infiltration of the tissues, which together with the edema produces alterations in the glands, the blood vessels and the stroma of the fibrous tissue. Microscopically, hyperesthetic rhinitis, as previously

shown by one of us in association with Fox,[17] displays the characteristics of chronic inflammation. In cases of early allergy the picture is that of simple hypertrophy, but after secondary infection occurs, hyperplasia develops. Changes in the epithelium, from the normal pseudostratified ciliated columnar type to a less specialized type of epithelium, take place.

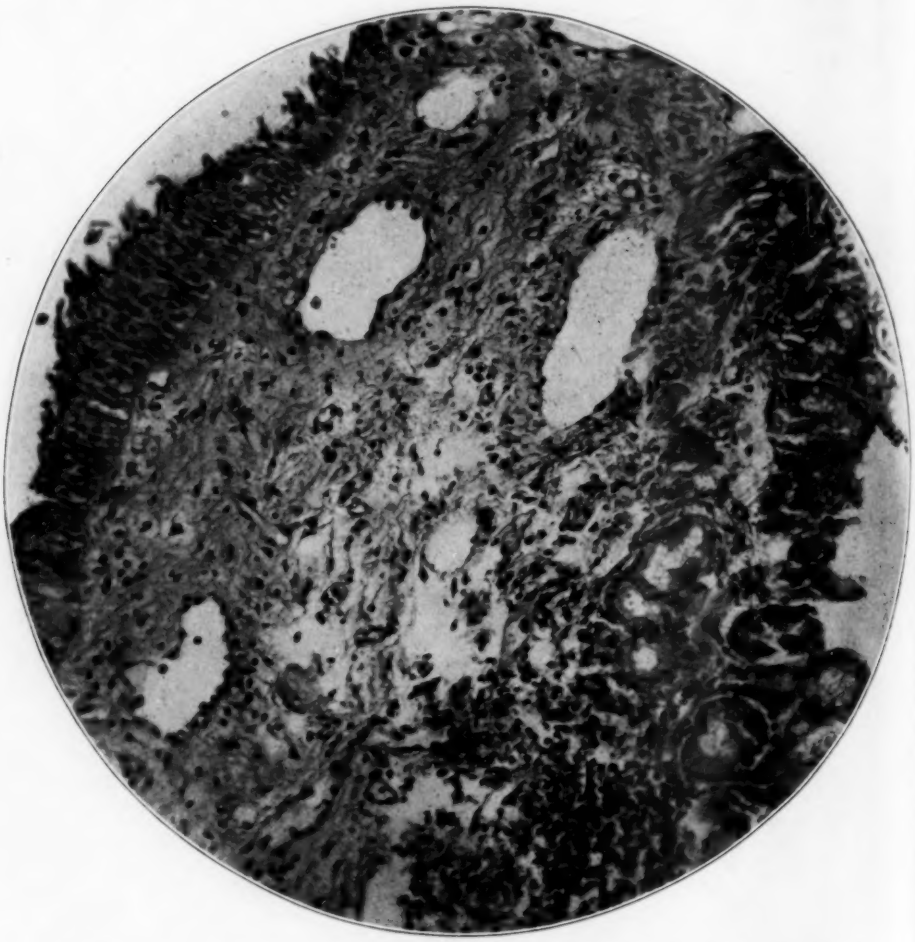


Fig. 5 (case 2).—First biopsy specimen of nasal mucosa taken after ionization. There is considerable desquamation of the lining epithelium. The stroma of the connective tissue contains a moderate round cell infiltration and numerous mucus-secreting glands and dilated lymph channels.

In some cases the epithelium is hardly thickened, while in others there is an increase in thickness and in cellular elements.

17. Fox, N., and Fabricant, N.: Hyperesthetic Rhinitis, Arch. Otolaryng. **18**: 181 (Aug.) 1933.

Hansel [18] and other workers have brought to our attention that in cases in which chronic infection is superimposed on an allergic process certain characteristic changes are produced which represent histologic pictures of both allergic and chronic inflammation of the infectious type. The two changes may vary in individual cases and even in parts of the

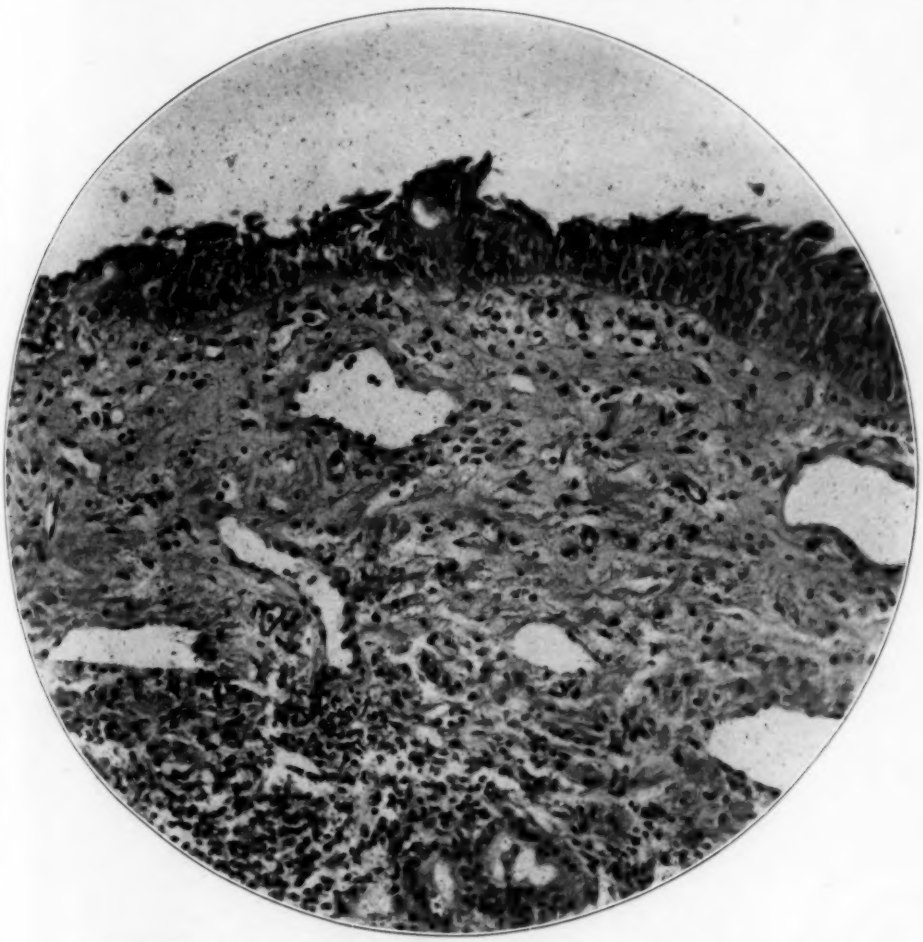


Fig. 6 (case 2).—Second biopsy specimen of nasal mucosa taken after ionization. The epithelium is of the ciliated columnar type. One portion shows slight desquamation; another part reveals one small area undergoing metaplastic change suggestive of stratified squamous epithelium. The stroma of the connective tissue is not cellular subepithelially. There is a periglandular round cell infiltration. Dilated lymph channels are to be seen.

18. Hansel, French K.: Allergy of the Nose and Paranasal Sinuses, St. Louis, C. V. Mosby Company, 1936.

same biopsy specimen. Thus, the allergic picture predominates in one case and the infectious in another, or there are equal or unequal representations of both. Hence, in one area the epithelium may be normal while in other areas it appears to be hyperplastic. The basement membrane is absent in one area, slightly thickened in another and markedly

Fig. 7 (case 2).—Specimen of nasal mucosa removed seven months after ionization (first biopsy). The epithelium is of the ciliated columnar type. The stroma shows a moderate round cell infiltration. The mucus-secreting glands appear to be unimpaired.

thickened in a third. In the subepithelial layer edema, eosinophilic infiltration and fibrosis vary in extent and concentration. The glands consequently are dilated or cystic, atrophic, compressed or fibrotic.

BIOPSY

In histologic studies made before as well as after nasal ionization of the human nose other workers have employed the biopsy method. In view of the fact that the vast majority of biopsy specimens have been obtained from either an allergic or a chronically diseased nose, it is

Fig. 8 (case 2).—Specimen of nasal mucosa removed seven months after ionization (second biopsy). The lining epithelium reveals moderate metaplasia suggestive of stratified squamous epithelium. One portion of the epithelium has undergone desquamation. A fair amount of fibrosis is present. There is a moderate round cell infiltration. (Structural differences are apparent between the first and the second specimen taken seven months after ionization, showing the danger of drawing faulty conclusions when the routine method of biopsy is employed.)

obvious that the mucosa had already undergone pathologic alterations. These alterations we have just mentioned. This problem presents itself: Does an allergic or a chronically infected nose afford a suitable "normal control" for the microscopic study of ionization? Dean and his associates [19] claimed that ionization of the human nose causes a definite

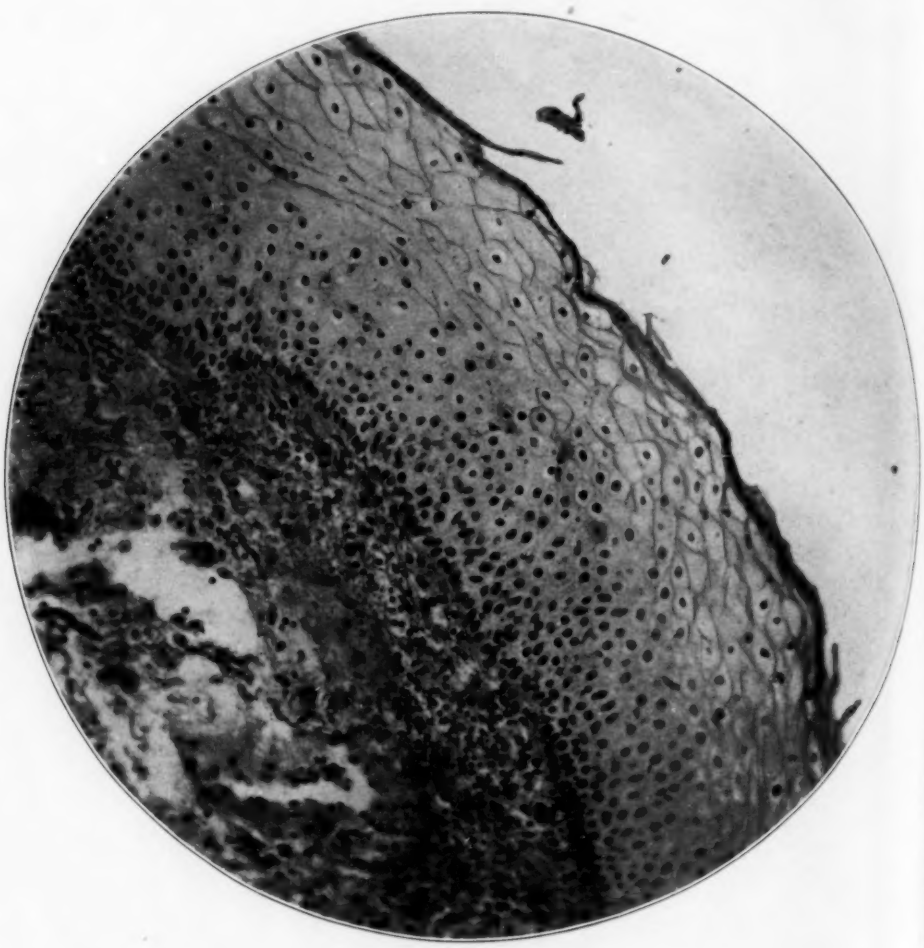


Fig. 9 (case 3).—Specimen of nasal mucosa taken before ionization. The epithelium is markedly thickened and shows pronounced change suggestive of stratified squamous cell epithelium. The stroma is fibrous and reveals a round cell infiltration.

19. Dean, L. W., and others: The Treatment of Allergic Rhinitis with Particular Reference to Ionization and the Control of Chronic Vasomotor Rhinitis, J. A. M. A. **108**:251 (Jan. 23) 1937.

pathologic process to arise in the mucosa. We have shown that this process is characteristic in chronic inflammation and in nasal allergy. It is therefore rational to believe that many of the changes seen microscopically after ionization are identical with those that existed before

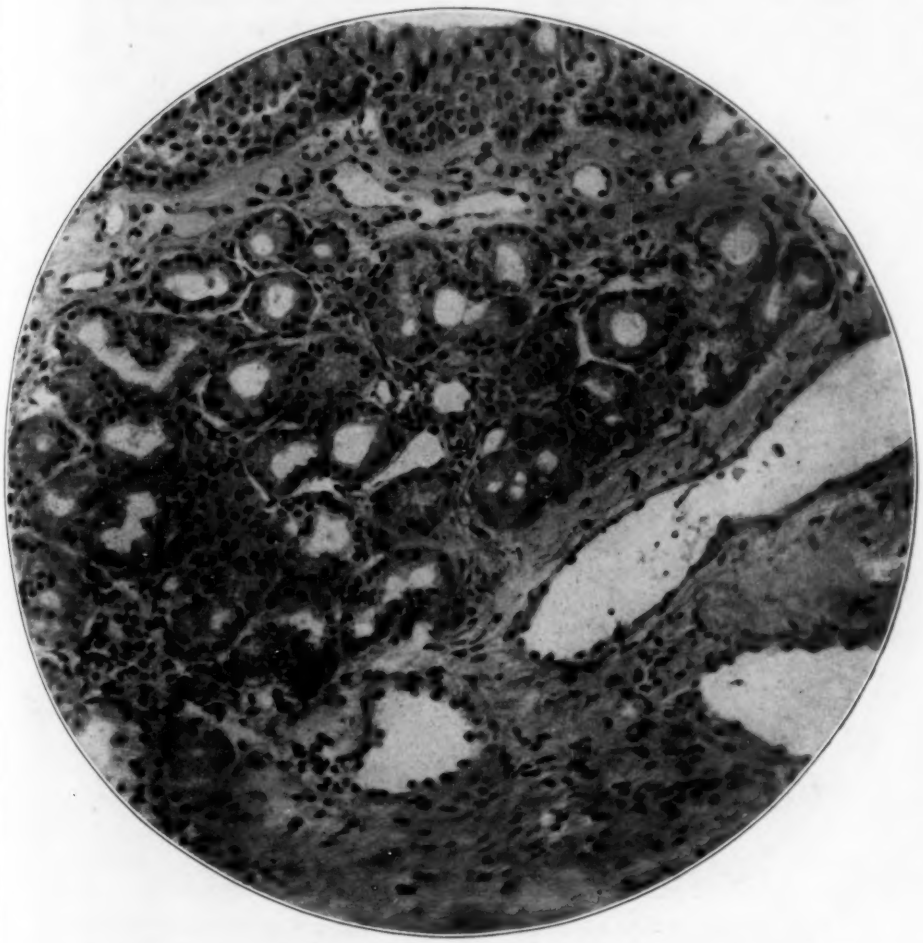


Fig. 10 (case 3).—Specimen of nasal mucosa taken after ionization. The epithelium is ciliated and columnar, although one large portion reveals some stratification suggestive of squamous cell epithelium. The stroma of the connective tissue shows numerous glands and dilated lymph channels, about which there is a round cell infiltration.

ionization. Dean and his associates have virtually recognized this problem, for they have written:

> In the human subject, probably because of the fact that we were dealing with chronic inflammation of the mucous membrane in the beginning, we have always found areas of persistent squamous cells following the ionization.

A specimen removed from a particular site before ionization may reveal microscopically relatively uninvolved and unaffected nasal mucosa; a specimen from a second site may show more pathologic involvement, while that from a third site may present highly altered nasal mucosa. Thus, two or more specimens taken before ionization frequently disclose great histologic variations in the tissue. Again, biopsy after ionization

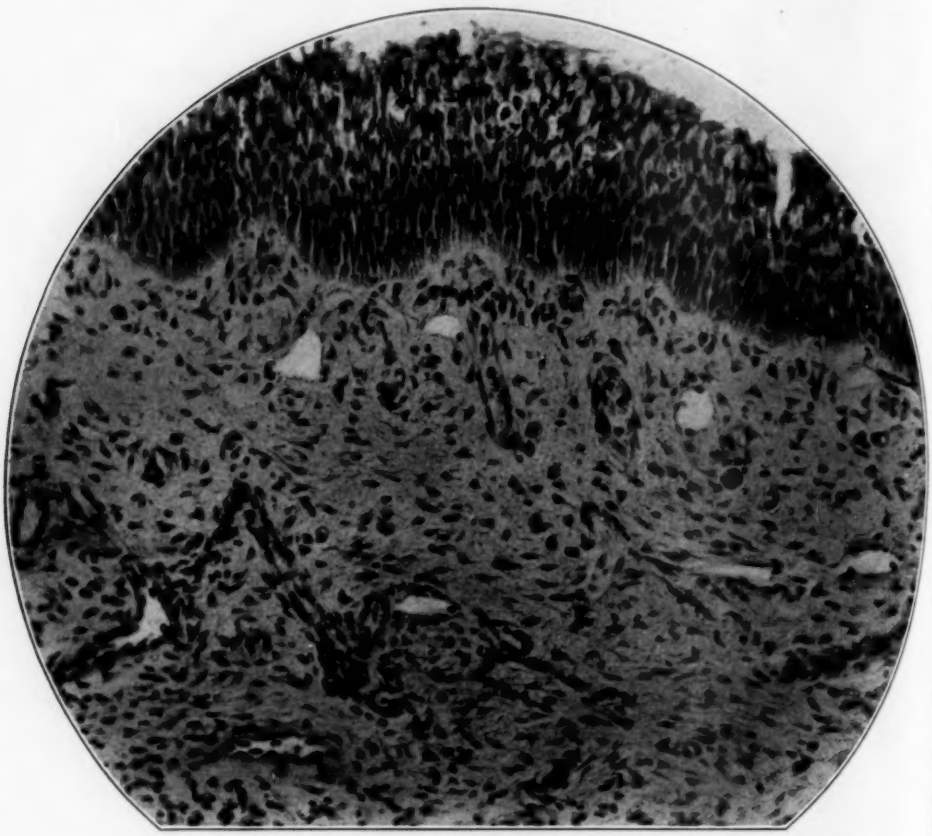


Fig. 11 (case 4).—Specimen of nasal mucosa taken nine years after ionization. The epithelium is pseudostratified. The stroma is fairly dense, with a moderately increased infiltration of small and large lymphocytes and occasional plasma cells and eosinophils. The mucus-secreting glands are normal.

occasionally reveals considerable epithelial insult and subepithelial change. On the other hand, according to our experience, not infrequently one observes less pathologic involvement than was seen in the microscopic section taken before ionization. Is it to be assumed that repair of the nasal mucosa has set in? Or are variable findings in one and the same turbinate to be allowed for?

Furthermore, if biopsy specimens from precisely the same site were removed before and after ionization, one would encounter destructive changes in the nasal mucosa produced by a punch or scissors which would render any accurate interpretation impossible.

It will be recalled that McMahon and Smit have studied the changes in the tissue of the mucosa of the nose and the sinuses of dogs and guinea pigs before and after ionization. The results of such studies are not altogether applicable to human beings. Nasal infection, allergy and environmental factors affecting man are not comparable to those affecting animals. McMahon established a method of study feasible in dogs but not in human beings. He was able to demonstrate sections of the nose and the sinuses in toto when the dogs were killed from eighteen hours to seven weeks after ionization. Until equally complete specimens are removed from the human nose, we contend that biopsy does not adequately represent the true condition both before and after ionization of the human nose. Smit asserted that he observed the formation of stratified squamous epithelium and its return to a normal ciliated columnar state when there was no chronic inflammatory process. This has not been our experience. Once normal pseudostratified epithelium has undergone definite transformation toward a stratified squamous form, there can be no reversion to the former normal state. Shields committed what is believed to be an error in assuming that allergic nasal mucosa could be interpreted in terms of the "norm." The work of Boling and Wenner on cats led to a different interpretation of the epithelial state from that advanced by McMahon and Smit as a result of their experiments on dogs and guinea pigs.

COMMENT ON PRESENT STUDY

We question the present method of histologic study of the nasal mucosa. It seems that all investigators (including ourselves) in attempting to evaluate the histologic observations after nasal zinc ionization have committed the same error. We believe that too often the state of the nasal mucosa prior to ionization, which was regarded as "normal," was in reality pathologic. Biopsy is too restricted to yield sufficient or adequate information about the entire nasal mucosa. Evaluation of the status of ionization with respect to vasomotor rhinitis and other types of nasal allergy must be obtained clinically rather than by microscopic investigation. The theoretical assumption that ionization produces harm in the nasal mucosa is not supported by histologic evidence. It can be asserted definitely that the electrocautery, drugs, chemicals, roentgen rays, radium, surgical trauma and the like produce far greater insult to the nasal mucosa than ionization.

SUMMARY AND CONCLUSIONS

Studies were made to determine whether the charge by certain authors that metallic ionization is harmful to the nasal mucosa is based on fact.

Careful histologic investigations have shown that fallacious deductions have resulted from the limitations of biopsy.

No histologic evidence has been produced to prove that nasal ionization is a harmful therapeutic procedure.

Until such a time as more conclusive histologic evidence of the effects of nasal ionization is available, evaluation of the procedure should be based solely on clinical observation.

DIRECT IMPLANTATION OF FREE NERVE GRAFTS BETWEEN FACIAL MUSCULATURE AND FACIAL TRUNK

FIRST CASE TO BE REPORTED

EDGAR P. CARDWELL, M.D.
NEWARK, N. J.

The application of free nerve grafts from a facial nerve trunk direct to the facial musculature has been done successfully for the first time, to my knowledge and so far as search of the literature reveals, in a case which I am reporting in the hope that the experience may be of benefit to surgeons and to their patients who have suffered considerable loss of the peripheral nerve.

S. K. was admitted to the Newark City Hospital on March 24, 1936, with a provisional diagnosis of tumor of the parotid gland. With the exception of the tumor, a mass measuring 7 by 5 cm., in front of and extending below the left ear, physical and complete laboratory examinations gave negative or irrelevant results. Roentgenograms made after the injection of iodized poppyseed oil 40 per cent into Stenson's duct showed a small amount of radiopaque material in the region of the mass. The normal ducts were not outlined.

On April 20 removal of the tumor with the atrophied remnants of the parotid gland disclosed such involvement of the facial nerve that it was necessary to divide the trunk proximally and all the small peripheral branches as they emerged from the tumor. Three peripheral fibrils were marked with black silk for subsequent identification, as also was the facial trunk.

Three days later, on April 23, under local infiltration with procaine hydrochloride, three branches of the external femoral cutaneous nerve were cut, dissected and marked with silk in preparation for grafting.

On April 27, a week after the removal of the tumor, the preauricular facial incision was reopened and the stump and two of the largest peripheral branches were identified. One marker had been lost. The two peripheral filaments identified had shrunk so that they were about the size of no. 2 Deknatel silk (0.012 inch [0.3 mm.]). The degenerated cutaneous branches from the anterior femoral cutaneous nerves were transplanted between the freshened end of the stump and the peripheral twigs. They were fastened to the stump by a single suture. The distal ends were laid parallel to and in close approximation with the marked peripheral twigs, without suturing. After short tunnels into the muscle plane of the face had been dissected, four fresh branches of the anterior femoral cutaneous nerve were also transplanted and laid out in these prepared channels. In other words, the proximal ends of all the free grafts were fastened to the facial stump by a single suture and molded into end to end apposition, and the distal ends were spread out fan fashion in the muscles of the face. A small rubber tissue drain

was left in the lower part of the incision for drainage of serum from the newly formed granulations which lined the wound. These granulations probably enhanced the chance of a take. The incision was closed with subcutaneous catgut, and skin clips and a moderate pressure bandage were applied.

On May 6 the patient was discharged, with the wound completely healed. Facial paralysis was still absolutely complete.

In November, clinical examination showed complete facial paralysis. Electrical stimulation, however, gave evidence of regeneration.

In May 1937, the face showed good function for both voluntary and emotional facial response in the lower two thirds and good closure of the eye. Clinically, the forehead showed no function.

The grafts used in this case averaged 6 cm.

Search of the literature disclosed a few cases of direct implantation of nerves into muscle with reinnervation of the muscle. The reports, for the most part, refer to experiments made on animals.

Nikolajew,[1] on the basis of his experiments, concluded: "The peripheral end of a motor nerve implanted into a previously uninnervated muscle heals in and by means of newly formed nerve endings it establishes a functional connection with the paralyzed muscle."

Stookey[2] found it possible to innervate peripheral muscles with a free nerve graft transplanted directly into the muscle and connected with a functioning nerve, without any apparent impairment of the function of the muscles normally supplied by the tapped nerve trunk. Stookey spoke of this graft as an artificial nerve branch. He suggested that his operation may be of great value in treating paralysis of an individual muscle resulting from anterior poliomyelitis.

Other experimental work along this line has been done by Erlacher,[3] Heineke[4] and Steindler.[5] Normal muscle brought into direct contact

1. Nikolajew, N. A.: Zur Frage der Implantation von Nerven in Muskeln (Nervenüberpflanzung auf Kehlkopfmuskeln), Monatsch. f. Ohrenh. **61**:923-937 (Aug.); 1005-1031 (Sept.) 1927.

2. Stookey, B. P.: Artificial Nerve Branches for Innervation of Paralyzed Muscles, Arch. Neurol. & Psychiat. **9**:249-254 (Feb.) 1923; Arch. Surg. **6**:731 (May) 1923.

3. Erlacher, P.: Einpflanzung des Nerven in den Muskel, Verhandl. d. deutsch. Gesellsch. f. Chir. **43**:194-195 (April) 1914.

4. Heineke, H.: Die direkte Einpflanzung des Nerven in den Muskel, Zentralbl. f. Chir. **41**:465-466 (March 14) 1914.

5. Steindler, A.: Direct Implantation of Motor Nerve upon Muscle Tissue (Neurotization): An Experimental and Clinical Study, J. Iowa M. Soc. **5**:436-439, 1915; Direct Neurotization of Paralyzed Muscles: Further Study of the Question of Direct Nerve Implantation, Am. J. Orthop. Surg. **14**:707-719 (Dec.) 1916.

with a paralyzed muscle will also innervate the paralyzed muscle. Successful results with this means of innervating muscle have been reported by various authors. Sheehan [6] employed pedicled strips from the temporal muscle to reanimate the facial muscles. Such a muscle graft carries with it nerve fibers which restore function to the basal muscle.

Drs. Wells P. Eagleton and Henry B. Orton gave suggestions and assistance in this work.

---

6. Sheehan, J. E.: Muscle Nerve Graft, S. Clin. North America **15**:471-482 (April) 1935.

CLOSURE OF PERSISTENT POSTOPERATIVE MASTOID FISTULA WITH A SUBCUTANEOUS PEDUNCULATED FLAP

LYMAN A. COPPS, M.D.
AND
G. L. McCORMICK, M.D.
MARSHFIELD, WIS.

There have been many methods presented for the closure of a persistent postauricular fistula following an operation on the mastoid. Some of these methods accomplish the closure of the defect in the skin but bridge over the deeper portion of the fistula, leaving a cavity, which may fill with secretion and cause the fistula to reopen. If the cavity can be obliterated completely and the skin closed tightly over it, it is more probable that healing will be complete. In order to accomplish this, Eagleton [1] filled the cavity with chips of bone and suggested the use of fat, Straatsma and Peer [2] used a free fat graft and Almour [3] employed a pedunculated flap from the temporal muscle.

In 4 cases we have used a method which has each time been successful and which may be recommended for its simplicity.

An incision is made in the scar of the wound through its whole length. This incision is carried down to bone and is made to encircle the fistula (fig. 1 *A*). Excessive scar tissue is excised, and all epithelium is removed from the fistula, which may extend into the antrum.

The anterior lip is undermined enough to provide a free margin for skin sutures (*a* of fig. 1 *B*). From the upper end of the incision a second incision (*b* of fig. 1 *B*), 2 or 2.5 cm. long, is made, backward and slightly upward. This incision is also down to bone. In a quadrilateral area (*c* of fig. 1 *B*), two sides of which have been outlined, the skin is dissected from the subcutaneous tissue, leaving the latter attached to the periosteum.

From the Marshfield Clinic.

1. Eagleton, W. P.: Reconstruction of Mastoid Cavity by Use of Bone Grafts and Chips, Laryngoscope **29**:272 (May) 1919.

2. Straatsma, C. R., and Peer, L. A.: Repair of Postauricular Fistula by Means of a Free Fat Graft, Arch. Otolaryng. **15**:620 (April) 1932.

3. Almour, R.: A Method for the Repair of Persistent Postauricular Openings, Laryngoscope **40**:799 (Nov.) 1930.

The flap of skin is held back with a retractor while a vertical incision (*a* of fig. 1 *C*) is made through the subcutaneous tissue and the periosteum downward to the bottom of the undermined area. The lower end of this incision (*a'* of fig. 1 *C*) is curved forward slightly to permit freedom of movement of the outlined subcutaneous flap. The width of this flap can be made to correspond with the size of the cavity it is to fill.

This flap is then dissected from the bone and swung forward to fill the fistula (fig. 2 *A*). If the posterior bony wall of this cavity is very high, it may be lowered by taking off some of the cortex with a

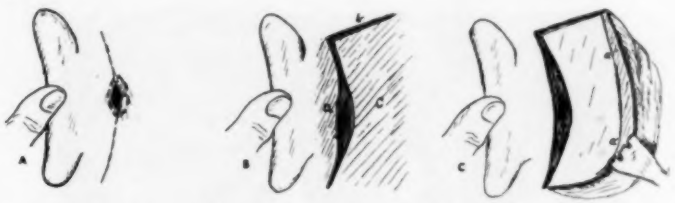


Fig. 1.—*A*, *B* and *C* indicate the first three steps in the operation.

Fig. 2.—*A*, *B* and *C* indicate the last three steps in the operation.

gouge, care being taken not to enter the diploe. The flap may be rather thin, and it may be made to fill the cavity best if the upper end is folded under (*a* of fig. 2 *A*, and fig. 2 *B*). It is held in place by a single catgut stitch, uniting it to the fascia at the upper anterior border of the fistula (*s* of fig. 2 *A*).

It will be found that the skin, which has been freed from the inelastic subcutaneous tissue, will stretch easily and that it can be approximated with mattress sutures and a tight closure made (fig. 2 *C*).

It is apparent that there is an area of bone posterior to the fistula which is covered only by skin. This, however, has led to no later ill effects.

Clinical Notes

LATERAL PRESSURE SPLINT FOR THE NOSE

V. H. Kazanjian, M.D., Boston

The pressure splint which I shall describe (figs. 1 and 2 *A*) provides a means of delivering a continuous, desired amount of lateral pressure at any desired point on the nose. It may be used to deliver pressure on opposite sides of the nose at the same time (fig. 2 *B*). Finally, it has one binding post which permits

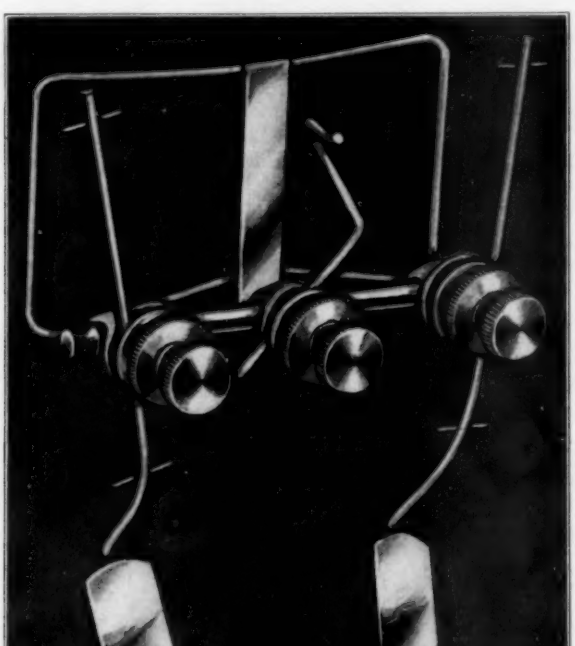


Fig. 1.—Drawing of the lateral pressure splint for the nose.

no "pendulum" action of its arm. The fixed arm from this post can be used in a number of desired ways, as an over the face point of attachment for traction bands, wires, etc.

The base is a simple wire frame fixed to the forehead, which is first taped and then covered with a layer of softened dental composition. The frame, which is embedded in this composition, will remain fixed to the skin for four or five days, its motion being limited at most to a few millimeters, that allowed by the elasticity of the skin.

The elastic bands used provide for the delivery of the force required. Their tension can be varied at will, and the slight shifts in the base and in the nose are immediately and automatically adjusted without any appreciable variance in the pressure applied to the nose. The point at which pressure is applied at the nose should be taped. A "stop" (fig. 2 *A*) will prevent overcorrection.

The apparatus which I have developed over a period of years has at least two advantages over similar devices in use at present: 1. The base at the forehead is simply secured and provides adequate stability. Plaster of paris headgear, while effective, has no inherent advantages and entails more labor and time in application. 2. The use of the elastic band for the sustained delivery of pressure at a desired point, while not a new principle, is frequently absent with other types of apparatus for pressure which the author has seen in use.

This apparatus was demonstrated at the meeting of the American Academy of Ophthalmology and Otolaryngology on Oct. 10 to 15, 1937.

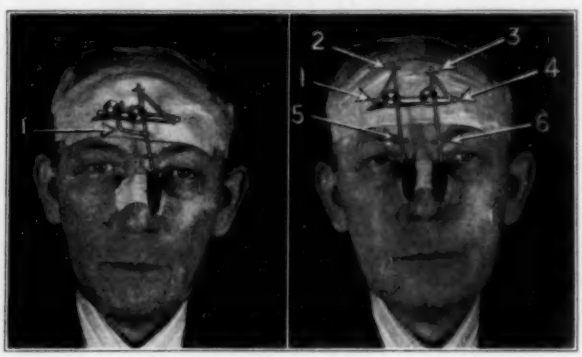


Fig. 2.—*A*, splint in position on one side of the nose. The stop, *1*, will prevent overcorrection. *B*, Splint in position to deliver pressure on both sides of the nose.

The indications for the use of this simple device will be outlined briefly: 1. It may be used in fresh fractures of the nasal bones, when it seems that lateral support is necessary to maintain the nasal bones in correct position after the usual methods of reduction. 2. A freshly reduced fracture which falls out of line within a day or two may be reduced again and the pressure splint applied. 3. In old fractures of the nasal bone, after the chisel, the mallet or some other means has been used to refracture the nose, the use of this splint is advisable to maintain reduction. With old fractures, contractures of soft tissue, etc., make the maintenance of reduction a difficult problem without some such apparatus.[1]

The base may be used, of course, with a fixed arm over the nose or the face. If it is used as such, it may be utilized for fractures of the nasal and of the malar bone and, in certain instances, for maxillary and mandibular fractures, when elastic bands may be attached to the fixed arm to secure upward tension such as that indicated on the facial bones.

1. My apparatus is manufactured by L. W. Falwell, Brighton, Mass. A patent has been applied for.

Progress in Otolaryngology

Summaries of the Bibliographic Material Available in the Field of Otolaryngology

---

TUBERCULOSIS OF THE UPPER PART OF THE RESPIRATORY TRACT

LITERATURE OF THE PAST TWO YEARS

GEORGE B. WOOD, M.D.
PHILADELPHIA

Recently the old debate regarding the possibility of a primary tuberculous infection of the larynx and the associated theories concerning the method of laryngeal invasion seems to have been reawakened.

Brieger and Pagel[1] report their findings in 4 cases in which the lesions at first looked very much like primary lesions of the larynx, perhaps of hematogenous origin. However, in 3 of the cases a careful clinical study showed demonstrable pulmonary lesions. In the other case it was only by careful postmortem examination that the pulmonary lesion was found. Histologic study of part of the pulmonary tissue finally revealed evidence of rupture of a small pulmonary nodule into a bronchus. As far as the pathogenesis of the laryngeal lesion is concerned, Brieger and Pagel say in conclusion: "We do not contest the possibility of an independent hematogenous development of a laryngeal lesion, but this should not be overrated."

Ruedi,[2] however, states that his experience of thirty years in the care of patients with laryngeal tuberculosis makes him feel that the laryngeal lesion is almost always of a lymphogenous or hematogenous origin. He goes further and states that the benign type of laryngeal lesions are lymphogenous and the severe or malignant types hematogenous. This belief is based on the variable occurrence of the disease, the fact that sputum with a positive reaction is not always associated with a laryngeal lesion, the preference of laryngeal tuberculosis to localize itself in the posterior commissure, due to a slowing up of the lymph stream in this locality, the belief that secondary infections of the middle ear and the nose should be considered as endogenic and the fact that tuberculous lesions of hematogenous origin occur in other portions of the body, such as the bones, the urogenital system and the serous membranes.

---

1. Brieger, E., and Pagel, W.: Papworth Research Bull. **1**:43-49, 1936.
2. Ruedi, T.: J. Laryng. & Otol. **52**:537-545 (Aug.) 1937.

McMahon,[3] in speaking of his experiences with tuberculous laryngitis at the Loomis Sanatorium, says that the larynx may become involved in two ways: first, by surface contamination from infectious exudate discharged from the lungs and, secondly, through the blood stream. He expresses the opinion that these two methods of infection can be differentiated clinically. The surface contamination tends to involve the intrinsic structures only, and the lesion is usually asymmetrical and is apt to occur on the side of the pulmonary involvement. This ipsilateral involvement, however, he cannot explain unless it is due to the fact that the patient tends to lie more on the affected side. When the lesion is hematogenous it is apt to be extrinsic, without ulceration and symmetrical.

(However, I still believe that the great majority of laryngeal infections are due to surface contamination, that lymphogenous infections practically never occur and that the hematogenous origin of laryngeal lesions must be limited to cases in which the tubercle bacilli are in the blood stream, as they are in miliary tuberculosis.)

A rather ingenious theory for the localization of the lesion in the larynx is advanced by Ringer.[4] He says that certain patients with pulmonary tuberculosis show a peculiar change in voice, having what he characterizes as the asthenic voice. This voice is due perhaps to general asthenia or merely to local weakening of the laryngeal muscles. It is typical of patients with pneumothorax, and, according to roentgen studies, it is most common in those with apical involvement plus localized pleural adhesions in the region of the clavicle. Also, there may be some interference with the recurrent nerve, either from pleural adhesions or from enlarged tracheal or tracheobronchial lymph nodes. The weakening of the chest muscles as well as of the laryngeal muscles themselves causes the patient to force voice production. This in turn produces congestion and hence a locus minoris resistentiae, so that the vocal cords and neighboring structures are most liable to become infected either by contact or through the blood.

Schuster [5] believes that the localization of tuberculosis in the larynx may be due to the fact that the parasympathetic reflex, spoken of by Pottinger, has afferent impulses running in the pulmonary branch of the vagus and efferent fibers in the laryngeal branch. He argues that irritation of the afferent fibers by inflamed pulmonary tissue produces trophic changes and atrophy in the larynx, which may lower the local tissue resistance on the side of the pulmonary lesion.

---

3. McMahon, B. T.: Am. Rev. Tuberc. **35**:109-116 (Jan.) 1937.

4. Ringer, M.: Monatschr. f. Ohrenh. **71**:132-137 (Feb.) 1937.

5. Schuster, F. P.: Observations on the Larynx in the Tuberculous, Arch. Otolaryng. **25**:23-36 (Jan.) 1937.

Hautant [6] has very timely called attention to the difficulty that he has experienced in certain fairly rare cases in making a differential diagnosis between malignant disease of the larynx and tuberculosis. He reports several illustrative cases. One patient, a man of about 60, examination of whose chest revealed only emphysema, had edematous vocal cords and subglottic granulation tissue clinically resembling carcinoma, which diagnosis was confirmed by biopsy. A hemilaryngectomy was carried out, and the specimen thus obtained showed on histologic examination numerous giant cells and other evidence of tuberculosis. There was proliferation of the basal cells of the epithelium, but it was not typical of an epitheliomatous process. (This is an extremely interesting case because, as pointed out by Manassa several years ago, when the subepithelial tubercle begins to invade the epithelium, the basal cells of the epithelium proliferate and may extend epithelial fingers deep into the surrounding tissue. Hence, if a cross section of the area were examined one might find epithelial nests that would suggest a malignant growth of the epithelium.)

Another patient was a man of about 50 who had been hoarse for a year or so but was otherwise in good health. There were complete fixation and swelling of the right hemilarynx, with an evident ventricular tumor. Temporary improvement followed cauterization, but three months later the condition became accentuated and Hautant felt that he was dealing with a malignant neoplasm. The patient died of acute phthisis. Contrariwise, in 2 other cases the diagnosis of tuberculosis had been made but the subsequent history proved the condition to be carcinoma. Hautant stresses the importance of not placing too much confidence on a single biopsy and says that several specimens should be taken from different parts of the laryngeal lesion. In every case in which a diagnosis of epithelioma seems obvious, a careful pulmonary examination should be made and taken into consideration. He expresses the opinion that a roentgenogram of the larynx can be a valuable aid in the differential diagnosis of cancer.

In considering the prognosis of laryngeal tuberculosis, Greene [7] says that the modern treatment undoubtedly makes it on the whole much more favorable. The important features of such treatment are rest, proper care of the associated pulmonary condition, employment when indicated of artificial pneumothorax and in well selected cases even of thoracoplasty, insistence on rest of the voice and judicious application of the electrocautery to the laryngeal lesion.

---

6. Hautant, A.: J. Laryng. & Otol. **52**:65-73 (Feb.) 1937.

7. Greene, J. B.: Prognosis in Laryngeal Tuberculosis, Arch. Otolaryng. **26**: 18-28 (July) 1937.

He says that the position of the lesion has an important bearing on the prognosis. As a rule, a lesion in the posterior commissure or on the vocal cords offers a more favorable prognosis than one on the epiglottis, the aryepiglottic folds or the arytenoid cartilages. Multiple lesions are of course more serious than single lesions, especially when they are bilateral. Perichondritis of the epiglottis or the arytenoid cartilage is a serious condition, while a pale edematous swelling in the latter region makes the prognosis almost hopeless. Further, he calls attention to the importance of the sedimentation rate in measuring the resistance of the patient to the infection. A rapid rate when associated with other evidences that the patient is on the downgrade is a most unfavorable sign. He does not suggest that the sedimentation rate is of any diagnostic value; he simply says that it is an evidence that the amount of tissue destruction has passed beyond the normal, making perhaps too great a load on the patient's recuperative processes. However, he has shown that in some cases in which there is a rapid sedimentation rate cauterization of the tuberculous lesion of the larynx is frequently followed by a marked improvement in the rate. This finding demonstrates that perhaps the increase in the rate may be due to the laryngeal lesion rather than to the pulmonary condition.

In the diagnosis of early lesions, Blegvad [8] says that the history of recurrent attacks of acute laryngitis is not of any value. According to his experience these recurrent attacks do not predispose to infection from the tubercle bacilli. He mentions the following conditions as indicative of early tuberculosis: First, isolated redness of a vocal cord is significant in a phthisical patient. Second, swelling and redness of the vocal process, with or without ulceration, is probably the most pathognomonic lesion. Third, prolapse of the ventricle of Morgagni, a swelling of the mucous membrane in the ventricle, which is seldom a true eversion, is frequently seen as an isolated lesion; in 34 of 48 cases typical laryngeal tuberculosis later developed. When this condition is associated with definite pulmonary tuberculosis it must be regarded almost always as a sure symptom of incipient laryngeal tuberculosis. Fourth, swelling of the lower surface of the vocal cords, although strongly suggestive of tuberculous invasion, is not quite as pathognomonic as prolapse of the ventricle or swelling of the vocal process. Fifth, swelling of the mucous membrane in the interarytenoid region is a frequent manifestation of laryngeal tuberculosis, and if ulceration is present the diagnosis is certain. For a more careful study of the posterior commissure, Blegvad recommends the use of the intralaryngeal mirror, which when introduced into a larynx treated with cocaine

8. Blegvad, N. R.; Burrell, L. S. T., and Thomson, St. C.: J. Laryng. & Otol. **52**:153-161 and 180-188 (March) 1937.

affords a good view of the whole of the posterior wall. Mirrors for examining the right and the left side can be used in the larynx. A red cushion-like swelling beneath the commissure is more commonly seen in patients with widespread tuberculosis of the larynx, but it may also appear as the first visible evidence.

Grain [9] has suggested the use of electrical anesthesia for the relief of pain in cases of laryngeal tuberculosis. This is essentially a medical anesthesia which eliminates in a localized area pain due to evolutive or avolutive lesions. The regional pains which form the extensive class of peripheral algias are divided into two categories, the truncular algias of the nerve tracts and the algias of the nerve endings. The electrical anesthesia affects only the algias of the nerve endings.

He obtains this anesthesia by iodide ionization, by encircling the larynx with special electrodes. The current may be supplied by a galvanic apparatus with a fine reducing coil working on 45 volt dry batteries or by accumulators capable of giving at least 15 milliamperes. The electrodes are made of unoxidable steel with a thickness of 0.5 mm. The anterior one is 75 mm. long and 35 mm. wide; the posterior one is 100 mm. long and 35 mm. wide. They are covered by several soft layers of absorbent cotton projecting at least 1 cm. beyond the edges. The anterior one is soaked with a 2 per cent solution of potassium iodide and linked with a negative pole. The posterior one is wet with pure water and connected with the positive pole. The anterior one is placed in front of the larynx and the posterior one at the nape of the neck, and they are held in place by a special collar of thick isolating rubber. From 10 to 12 milliamperes of current is used. The sittings last half an hour or longer and are repeated daily until the duration of the anesthesia reaches twenty-four hours; they are then repeated only when the pain reappears. During the treatment the patient usually has a sensation of stricture in the neck and a copperish taste. The sensation of stricture disappears in four or five minutes, while the taste and salivation may persist during the whole treatment.

Grain claims that this anesthesia is instantaneous and total, regular in its onset, and progressive in that after each treatment it persists for a longer time. It is stable and not influenced by the extent of the lesion, but the stability is not insured if a local anesthetic is applied. The treatment, however, must be considered only as a systematic treatment of pain, for it has no curative value. As it acts only on the diseased nerve endings, there is no suppression of the tactile sensitiveness of the mucosa, and hence it does not predispose the patient to the inspiration of food.

9. Grain, R.: Tubercle **17**:261-266 (March) 1936.

While I am still on the subject of the relief of pain caused by tuberculous lesions of the larynx, I find it interesting to note that according to Stott [10] a large part of the pain is due to pressure on the sensitive lymph nodes of the neck by the surrounding muscles. He proposes to block not only the sensory branches of the superiór laryngeal nerve but the motor nerves that supply the posterior belly of the digastric muscle and the mylohyoid muscle. The blocking of the branch of the facial nerve that runs to the posterior belly of the digastric muscle can be done immediately after the blocking of the superior laryngeal nerve is finished. He simply partially withdraws the needle and by depressing the barrel of the syringe is able to deposit a few drops of the anesthetic near the lower edge of the posterior belly of the digastric muscle without making a fresh puncture in the skin. The mylohyoid nerve, which supplies the anterior belly of the digastric muscle and the mylohyoid muscle, is reached through the mouth by a technic somewhat similar to that used in dental practice to reach the inferior dental nerve, but the level is suitably lower. Scott says that injections may be made on both sides if necessary. The anesthetic usually recommended is a solution of procaine hydrochloride in from 60 to 80 per cent alcohol with a little chloroform. The injections are seldom required more than once a week. He claims that this blocking of the branch of the facial nerve that runs to the digastric and the mylohyoid nerve does not cause pharyngeal paralysis, as the glossopharyngeus, the spinal accessory and the hypoglossal nerves are not affected.

When it comes to the curative treatment of laryngeal tuberculosis, it is apparently universally recognized that the use of the electrocautery is by far the most important method of combating the disease. There is some variance of opinion as to the indications, and a number of authors apparently use this instrument only as a last resort. For instance, Ruedi [2] avoids operative intervention when there is no pain, when the lesion is more or less localized or when the pulmonary condition is still active. In such cases he suggests roentgen therapy or the intravenous use of solganol B (the disodium salt of 4-sulfomethylamino-2-auromercaptobenzene-1-sulfonic acid). He advises against the use of general treatment with an arc lamp. He is apparently energetic in his use of the cautery, having devised a spiral platinum burner of various shapes and sizes, which he employs at almost white heat. He says that in certain cases it is necessary to destroy the false vocal cord right up to the ventricle of Morgagni. (Personally, I believe that widespread cauterization of the ventricular band may lead to disastrous results. In 1 case recently in which this was attempted in order to overcome the

10. Stott, L. B.: Brit. J. Tuberc. **30**:155-159 (July) 1936.

stenosis caused by the infiltration in the false cord, there was a severe reaction, with edema so extensive that tracheotomy was necessary and the patient had to be fed for several weeks through a nasal tube.)

Blegvad [8] prefers ultraviolet radiation with a quartz lamp for the conservative treatment of patients with laryngeal tuberculosis and says they are especially applicable in the case of those who are very sick. However, if improvement is not shown within a few months, operative work by the indirect method is indicated. At the Finsen Institute between 1922 and 1936 there were performed 401 excisions, 527 galvanocauterizations, 40 amputations of the epiglottis, 127 injections of alcohol and 49 resections of the superior laryngeal nerve. By these procedures 29.1 per cent of the patients were cured, 10.2 per cent improved and 60.7 per cent unimproved.

Schuster [8] insists on absolute rest of the voice, his patients not being allowed even to whisper. Although the use of local anesthesia, the injection of alcohol into the superior laryngeal nerve and the reflection of sunlight into the larynx may be useful as conservative measures for the relief of pain, he says that the galvanocautery is the most valuable agent. He stresses the point that no attempt should be made to destroy all of the diseased tissue by the cautery, the aim being only to stimulate the healing process. He calls attention to the fact that collapse of the lung by pneumothorax, phrenic evulsion or even thoracoplasty is often valuable as a curative agent but is much more useful in the prevention of the laryngeal involvement.

In regard to the use of ultraviolet rays, Kowler and d'Halluin [11] state that the favorable results of heliotherapy have been at least exaggerated. Even when ultraviolet rays are combined with other types of rays, they say that the ultraviolet ray has slight curative action. They express the hope that future research will provide more knowledge concerning the action of the ultraviolet ray as it is found in the atmosphere, because at present there is little evidence to show that the violet ray is of much importance in the treatment of laryngeal tuberculosis.

Rubaltelli,[12] after reviewing the literature on the treatment of tuberculous laryngitis, compares the results obtained with periarterial sympathectomy and roentgen treatment. Each method seems to produce a reflex intralaryngeal vasodilatation. Except for the fact that each seems to control the painful dysphagia, the results reported in the literature as to their curative effect on infiltration and ulceration are somewhat conflicting. The advantage of roentgen therapy is that it can be used in all cases regardless of both the local and the general condition of the patient. Rubaltelli says that it gives more lasting improvement than

11. Kowler and d'Halluin, M.: Ann. d'oto-laryng., March 1936, pp. 281-284.

12. Rubaltelli, E.: Oto-rino-laring. ital. **6**:558-562 (Dec.) 1936.

does sympathectomy and that when the proper dose and the proper technic are used there are few, if any, accidents or complications. He strongly advises against any prolonged or bloody operations on tuberculous patients if they can possibly be avoided.

Furman [13] has made a rather complete review of the literature of so-called primary tuberculosis of the palate, tonsils and adjacent regions and reports an interesting case of his own in which he successfully treated the patient with 50 per cent trichloroacetic acid. This patient had no clinical evidence of pulmonary disease, and the discomfort in his throat had started about six months before he was seen by Furman. When he was first seen there was an extensive dirty-gray ulceration, with punched-out verrucous margins, on both tonsils, the anterior and posterior pillars, the soft palate, the uvula and the posterior pharyngeal wall. The ulcers on the tonsils were deep and surrounded by large masses of infiltration. The palatal ulcers were superficial, while those on the pharyngeal wall were moderately deep and surrounded by red edematous nodules. The hypopharynx was not involved, nor was the larynx, epiglottis or base of the tongue, and nasal involvement was not evident. Smears from the ulcers showed Vincent's organisms. The diagnosis was established by biopsies of material taken from the edge of the ulcers. These showed tuberculous granuloma, with typical tubercle and giant cells, and the presence of tubercle bacilli was demonstrated both by staining and by inoculations of guinea pigs.

Lactic acid was used at first but there was no improvement, and then 50 per cent trichloroacetic acid was swabbed over the whole area involved. This caused a blanching of the tissue but not cauterization, as the blanched appearance disappeared in twenty-four hours. The ulcers immediately began to heal, and the infiltrations disappeared gradually. Treatments were given every second or third day for three months, at the end of which time the ulceration was completely healed, though there were some areas of infiltration. Four months after the first visit the tonsils were removed, with the aid of local anesthesia, and histologic examination showed tuberculosis. After further applications of trichloroacetic acid the remaining infiltration of the pharynx completely disappeared, as well as the swelling and tenderness of the cervical lymph nodes.

Roentgenograms of the chest were taken in the second month after treatment was started and again in the twelfth month. The report was as follows: "The lung markings show no evidence of active acid-fast pulmonic involvement nor foci of infiltration or consolidation. There are, however, numerous calcified nodes opposite both hila and several calcified tubercles near the right base." (Primary tuberculosis of the tonsils

13. Furman, A.: Ann. Otol., Rhin. & Laryng. **46**:456-470 (June) 1937.

is not very rare, and it is perfectly conceivable that the palate and the neighboring portions of the pharynx might have been involved by direct extension of the disease from the tonsils. However, the finding of calcified tubercles near the base of the right lung would make it impossible to class the condition beyond doubt as primary tuberculosis of the faucial region.)

Myerson [14] observed 708 cases of laryngeal tuberculosis during the years 1932, 1933 and 1934 and performed tracheotomy for obstructive lesions in 9. In 2 other cases of pulmonary tuberculosis without specific laryngeal involvement tracheotomy was required, in one because of associated carcinoma and in the other because of bilateral paralysis of the abductor muscles. In a large majority of the other cases the laryngeal stenosis was due to fixation of the vocal cords in the median line or almost in the median line by a lack of motility of the cricoarytenoid joint. As to the ability of the patient to get rid of mucus and exudate from the bronchi after tracheotomy, he says that some of the patients found expulsion more difficult after tracheotomy while others found it easier. As to the probability of the tracheotomy wound's becoming infected, he says that infection depends entirely on the amount of sputum and the pyogenic organisms therein rather than the presence of a few tubercle bacilli. Of the 11 patients operated on, 3 had infection of the wound, and of those who did not have an infected wound 5 had sputum with a positive reaction.

Tracheotomy is not to be considered as a curative measure but should be done only to relieve obstruction which cannot be eliminated by endolaryngeal instrumentation. However, patients who have a partially obstructed laryngeal airway must be watched carefully, because fixation of the cricoarytenoid joint may develop at any time. (The only criticism I would have of Myerson's findings is that paralysis of the laryngeal nerve probably occurs more frequently in cases of advanced pulmonary disease than is generally supposed. It is possible that in some cases so-called fixation of the arytenoid cartilage may be paralysis. However, this possibility does not change the indications for tracheotomy.)

Putnam [15] reports a case in which infraglottic perforation followed tuberculosis of the larynx. The patient was a white man 59 years of age who had an external abscess in the front of the neck. The abscess was opened anterior to the thyroid cartilage and 90 cc. of pus drained. This pus was laden with tubercle bacilli. The patient died a little over a month later, with general miliary tuberculosis. At autopsy a large ulceration was observed in the infraglottic region; it extended from

14. Myerson, M. C.: Tuberculosis of the Larynx Requiring Tracheotomy, Arch. Otolaryng. **23**:1-17 (Jan.) 1936.

15. Putnam, F. I.: Am. Rev. Tuberc. **33**:75-77 (Jan.) 1936.

the cords to the first tracheal ring. The mucosa was completely destroyed, the cartilage was necrosed and on the left side near the posterior wall a perforation was seen. In his summary Putnam says that 8 cases of perforation of the larynx by a tuberculous process had been reported between 1836 and 1927.

Tuberculosis of the trachea and the main bronchi has apparently been receiving more attention recently than has been accorded to it for some time. Kernan [16] stresses the use of the bronchoscope both for diagnosis and for treatment. The disease may be made manifest by the presence of tuberculoma, ulceration or varying degrees of disease of the bronchus from scar formation. As far as treatment is concerned, he says that the ulceration should be coagulated, touched with nitrate of silver or irradiated with specially designed quartz rods. Strictures should be dilated with copper ionization and tuberculomas removed with forceps, the base being coagulated immediately.

Moorhead [17] reports 2 cases of tuberculosis of the bronchi, 1 in an infant 17 months old. A localized tuberculoma was present, which was removed through the bronchoscope, and the area was treated with high voltage roentgen therapy.

Lewy [18] reports an interesting case in a 30 year old white woman who had had a nontoxic enlargement of the thyroid for eight years, with absence of radial and brachial pulsations from the left arm. At the time of operation on the thyroid the second portion of the subclavian artery was found to be obliterated by dense connective tissue. Four months after the operation marked dyspnea developed, and bronchoscopic examination showed a mass about the size of a marble bulging into the lumen of the trachea just above the carina. Biopsy showed this mass to be caused by fibrocaseous tuberculosis. She died three months later, and at autopsy there was observed a firm, dense, grayish white area of scar tissue extending to the left of the aortic arch and into the supraclavicular region. It connected with the dense scar which followed the trachea down to the left bronchus. In the scar were a number of discrete caseous areas. An irregular ulceration in the lower portion on the left side of the trachea extended for a short distance into the left bronchus. Several rings of the trachea were exposed, and a perforation passed into the surrounding scar tissue. The tissue from the base of the ulceration showed cellular and vascular tissue, with nodules of epithelioid and giant cells. There was an encapsulated lesion in the lower lobe of the right lung, which was probably the primary infection.

16. Kernan, J. D.: Ann. Otol., Rhin. & Laryng. **46**:763-770 (Sept.) 1937.
17. Moorhead, R. L.: Ann. Otol., Rhin. & Laryng. **46**:754-756 (Sept.) 1937.
18. Lewy, R. B.: Ann. Otol., Rhin. & Laryng. **46**:735-739 (Sept.) 1937.

There still are, apparently, some capable otolaryngologists who do not consider the presence of pulmonary tuberculosis a contraindication for tonsillectomy. Pinkerton [19] during the past five years has done 102 tonsillectomies on adults, with local anesthesia, and 40 on children, with general anesthesia. These were tuberculous patients. None of them were operated on until they had been thoroughly studied by the medical department. Careful consideration was given to the extent of the pulmonary lesion, to its trend toward healing or progression and its activity as evidenced in recent roentgen examination, study of the blood and sputum and consideration of the type and extent of the tuberculous complications. If in the opinion of the medical department these factors did not contraindicate the operation and the tonsils appeared to be an obstacle in the way of recovery, the operation was done at the earliest opportunity but with special preparation and technic. When the anesthetic used was local, operation was done by blunt dissection and snare, bleeding being controlled by suture when necessary, and the tonsillar fossae were painted once a day postoperatively with compound tincture of benzoin plus 1 grain (0.064 Gm.) of mercury bichloride to the ounce (30 cc.). Thirty of the 63 adult patients but only 2.5 per cent of the children had tuberculous tonsils. Those whose sputum had a positive reaction had a much higher percentage of tuberculous lesions in the tonsils, though such lesions were observed in the tonsils of 5 of the 34 patients whose sputum had a negative reaction. Two patients died, one, a young girl, seventy-five days after the operation, apparently because of heart failure. The other, classified as a patient with far advanced tuberculosis whose sputum had a positive reaction, proved at autopsy to have a dermoid cyst of the lung, with no evidence of tuberculosis.

Pinkerton expressed the opinion that in properly selected cases of tuberculosis there is no inherent danger and that tonsillectomy may be of considerable benefit in speeding convalescence. However, there is no special mention made of his results with the 40 children operated on under general anesthesia. He says that the fact of frequent infection of the tonsils in cases of pulmonary tuberculosis is a further indication for their removal, because removal eradicates a certain amount of the tuberculous disease. He does not believe in the use of the cautery for tuberculous tonsils, preferring their radical removal. (I still believe that the surgical removal of tonsils in a case of active pulmonary tuberculosis is scarcely ever justifiable and that tuberculosis of the tonsils can be treated successfully by cauterization.)

Two interesting cases of tuberculous tonsils are reported by Leubner.[20] The first patient was a 17 year old girl who at the time of

19. Pinkerton, F. J.: Tr. Am. Laryng., Rhin. & Otol. Soc. **41**:540-552, 1935.

20. Leubner, H.: Klin. Wchnschr. **16**:779-783 (May) 1937.

the examination was almost cachectic. She had a history of numerous infections of the teeth, mouth and eyes, a temperature of about 37 or 38 C. (98.6 or 100.04 F.) marked cervical adenopathy and large pedunculated tonsils, the crypts of which were filled with débris. Though the chest was clinically clear, the roentgenogram showed old tubercles in the apex of the left lung. There was a marked increase in the sedimentation time, and a blood count showed red cells 2,240,000, hemoglobin 61 per cent and white cells 3,000, with a nuclear shift to the left. Although the condition appeared possibly to be miliary tuberculosis, the tonsils were regarded as a point of entrance and removed. The results of the operation were apparently beneficial; the patient gradually became afebrile and felt well. The temperature rose again, however, about two weeks after the operation, at which time the cervical lymph nodes were excised. The patient again improved, so that six weeks after the tonsillectomy she could hardly be recognized as the same person. On histologic examination both tonsils showed extensive tuberculosis. In the second case, however, the results were quite different. A 20 year old girl had been perfectly well up to the time of a second attack of tonsillitis, but after this attack she had marked cervical adenopathy, a slight rise in temperature and general malaise, but no evidence of any organic lesion other than diseased tonsils. The tonsils were removed, but after the operation tuberculous pleurisy developed, the final outcome of which was not stated. On histologic examination the tonsils were found to be tuberculous.

Krishnaswamy, Goyle and Vasudevan [21] report the histologic examination of tonsils removed in 2,477 cases. There was evidence of tuberculosis in 15, or 0.6 per cent of all the cases. However, they quote from Mullin, who in 1923 reported the examination of tonsils removed in 400 cases. In 4.25 per cent tuberculosis was present. The former authors state that in no case in which they saw histologic evidence of tuberculosis could the disease be recognized by clinical examination. Though sometimes the tubercles showed a tendency to coalesce, caseation was uncommon. In most cases proliferation of the reticular cells and the formation of fibroblasts was pronounced. These authors acknowledge but are unable to explain the fact that tonsillar tissue shows a marked resistance to caseation, as is so frequently evidenced in tuberculosis of the lymph nodes. They say that histologic evidence indicates that the ultimate tendency of the tuberculous lesion in the tonsil is toward recovery and that the widespread fibrosis which is sometimes seen in tonsils may be an extinct tuberculous infection.

21. Krishnaswamy, K. G.; Goyle, A. N., and Vasudevan, A.: Indian M. Gaz. **71**:254-258 (May) 1936.

Abstracts from Current Literature

Ear

THE VASOMOTOR CONTROL OF THE EAR. P. FRAZER, J. Laryng. & Otol. **51**:579 (Sept.) 1936.

Frazer investigated the path taken by the vasoconstrictor impulses to reach the middle and the internal ear by means of a special dye injection technic, using the cat as the experimental animal. He found that stimulation of the stellate ganglion gives rise to vasoconstriction in the external, the middle and the internal ear. The same result is also obtained, as far as the ear is concerned, when the superior cervical sympathetic ganglion or the cervical portion of the sympathetic trunk is stimulated. These results are unaffected by decortication of the internal and external carotid arteries. No vasoconstriction occurs anywhere in the ear when one stimulates the stellate ganglion after dividing the cervical portion of the sympathetic trunk, thus excluding the possibility that vasoconstrictor impulses will reach the ear from the stellate ganglion by way of the periarterial plexus of the vertebral artery. An experiment is described which suggests that the vasoconstrictor fibers destined for the area supplied by the first and second divisions of the trigeminal nerve pass through the tympanic plexus of the middle ear. The bearing of this work on cervical sympathectomy for aural conditions is discussed.

EDITOR'S ABSTRACT.

REPORT ON EIGHT HUNDRED AND NINETY-ONE CONSECUTIVE CASES OF ACUTE MIDDLE-EAR SUPPURATION AND MASTOIDITIS WITH INTRACRANIAL COMPLICATIONS IN ONE HUNDRED AND THIRTY-NINE CASES DURING THE PERIOD OF 1920 to 1934. J. S. FRASER and G. C. HALLIDAY, J. Laryng. & Otol. **51**:619 (Oct.) 1936.

Fraser and Halliday deal with 891 cases, 60 of which were fatal; intracranial complications were present in almost 15 per cent, of which 6 per cent were fatal. The authors included cases in which the patient was admitted in a comatose condition due to meningitis, because one of the main objects of their study was to discover, if possible, the mortality rate for acute suppurative otitis media and its complications. However, they excluded cases of tuberculous otitis media and cases of tuberculous meningitis, as these are not acute suppurative conditions. In connection with the intracranial complications, the authors found that many patients died of purulent meningitis and that there was little evidence of abscess in the tympanic antrum and air cells and no sign, either at operation or at necropsy, of extradural abscess or other intracranial complications. Further, postmortem examination did not show any evidence of infection of the paranasal air sinuses. The meningitis from which 1 patient died had little or no connection with the acute otitis media; postmortem examination showed that the spleen was enlarged to two or three times its normal size. The meningitis appeared to be due to an infection of the blood. It may be that in the presence of otitis media the micro-organisms circulating in the blood tend to settle in the meninges and give rise to meningitis. It was noted in several cases that the meningitis was most marked over the vertex, whereas in those cases in which there was well marked disease of the mastoid air cell, with extradural abscess or septic thrombosis of the lateral sinus, the meningitis was most marked at the base. The authors found that the transverse labyrinthine drainage of West and Scott is useless in the treatment of meningitis except when the meningitis is secondary to labyrinthitis. Blood cultures proved disappointing. They are of value only when positive. Further, if the patient is very ill the surgeon has usually to decide

the question of operation before the report of the culture is received. Like other otologists, the authors have been struck by the frequency of the great enlargement of the spleen in cases of septicemia, i. e., infection of the blood without protective thrombosis in the lateral sinus. They agree, on the whole, with the modern teaching that an abscess of the brain should not be operated on in the early stage, that is, that the surgeon should not interfere in the stage of red softening but should wait for the formation of a more or less definite encapsulated abscess. Unfortunately, it is not always possible to follow this rule, especially in cases of cerebellar abscess.

EDITOR'S ABSTRACT.

CHANGES IN THE LABYRINTHINE CAPSULE OF THE RABBIT IN LEAD POISONING. W. KRAINZ, Monatschr. f. Ohrenh. **70**:1211 (Oct.) 1936.

Krainz says that the subcutaneous injection of an aqueous solution of lead acetate produced in the skeletons of rabbits changes like those that are associated with acute osteoporosis. The temporal bones of the rabbits were subjected to an especially careful examination, because it was to be determined how the temporal bone and particularly the labyrinthine capsule react to the lead poisoning. It was found that in the temporal bone, as in the other portions of the skeleton, the processes of transformation, particularly osteoclasis, were pathologically increased, the periosteal as well as the endochondral capsule being involved. The changes were especially severe in the endochondral capsule of the semicircular canals, whereas the changes in the endochondral capsule of the cochlea were slight. The apposition of new bone was much less pronounced than the destruction of bone. The author thinks that the osteoporotic changes in the labyrinthine capsule, like those in the skeleton, are caused by the acidosis of lead poisoning. As the result of this acidosis, mineral salts, particularly calcium salts, are withdrawn from the organism.

EDITOR'S ABSTRACT.

TYPICAL AND CONSERVATIVE RADICAL OPERATION FOR OTITIS MEDIA. E. RUTTIN, Monatschr. f. Ohrenh. **70**:1217 (Oct.) 1936.

Ruttin discusses the typical radical operation only in general, because he has not yet organized his large material. However, he summarizes the 82 cases in which he performed the conservative radical operation. Depending on the site of perforation, he differentiates five types of otitis. After discussing these five types, he takes up the surgical aspects of the 82 cases. Then he takes up the question of whether the conservative radical operation effects cure more readily when cholesteatoma is present or when it is not. He found that of 28 patients who were cured, 12 had cholesteatoma and 13 had not; in the other 3 cases the surgical report did not state whether cholesteatoma was present. After describing the results of the typical and of the conservative radical operation as they relate to operative cavity, he points out that relapses are much less frequent after the conservative method than after the radical method. Further, he takes up the functional results, pointing out that if the right patients are selected for the conservative radical operation, the functional results are generally favorable. Regarding the technic of this intervention he says that on the whole it is much like that of the typical radical operation, except that he preserves the tympanic membrane and the auditory ossicles; that is, he removes the posterior wall of the auditory meatus and also the bridge and the attic wall, as suggested by Bondy. The intervention is of course adapted to conditions in the individual cases.

EDITOR'S ABSTRACT.

Pharynx

THE SURGICAL TREATMENT OF PERITONSILLAR INFLAMMATIONS. R. SCHROEDER, J. Laryng. & Otol. **51**:574 (Sept.) 1936.

Schroeder states that peritonsillar abscess is usually a benign though painful disease, but in the few cases in which complications occur it must be considered dangerous; a more radical surgical treatment is therefore justified. During recent

years the surgical treatment has been partial or total tonsillectomy. The question of tonsillectomy as a treatment for peritonsillar abscess is still under discussion. Many surgeons agree on the necessity for tonsillectomy "à chaud" in certain cases. Those who do not share this opinion give as a reason that they think the procedure too severe, as most abscesses can be opened simply by incision, and they do not find the operation on inflamed tissue an efficient surgical procedure. The fact that several authors have reported fatal complications after tonsillectomy for acute peritonsillitis shows the danger of this treatment. To reduce the risk, others have suggested detachment of the upper pole of the tonsil and partial tonsillectomy, i. e., cutting away the upper pole of the tonsil or making a large opening to the tonsillar bed through the anterior faucial pillar. Generally the abscess is behind the upper pole of the tonsil, is conveniently opened by incision at the top of the anterior faucial pillar and is well drained by this proceeding. When the abscess occurs behind the lower half of the tonsil no pus will appear with the incision previously described, and, as experience shows that the lower abscess gives rise to severe complications, such as thrombosis of the internal jugular vein and septic embolism, it is of great importance that it should be efficiently opened. Further surgical treatment is necessary. At the author's clinic 56 patients with peritonsillar abscess were treated as soon as possible by detachment of the upper pole of the tonsil, with partial or total tonsillectomy, in order to get complete evacuation of the pus. The indications for further surgical treatment were incomplete evacuation of the pus after incision, nephritis, oedema laryngis, thrombophlebitis, septic conditions and parapharyngeal abscess. The operations were all performed with the patient under local anesthesia, which has, among other advantages, that of stopping the trismus. The result of these procedures is good, and it is not proved that the procedure gives a less satisfactory postoperative course than incision. It is remarkable how many cases of peritonsillar inflammation are complicated by parapharyngeal abscess.

EDITOR'S ABSTRACT.

SOME REMARKS ON SUPPURATION IN THE PARAPHARYNGEAL SPACE. R. SCHROEDER, J. Laryng. & Otol. **51**:631 (Oct.) 1936.

In treating a series of patients with complicated peritonsillar abscesses, Schroeder observed not only the different ways in which the pus can travel when the pressure in the inflamed tonsillar bed is getting too high but the different clinical pictures presented. He mentions only the suppurations in the parapharyngeal space caused by tonsillar or peritonsillar inflammation, all suppurations of other kinds being disregarded. Following a description of the anatomy of this region, the author says that, according to his observations, the clinical picture of abscesses in the parapharyngeal space depends on which place in the tonsillar bed the pus has broken through. Parapharyngeal suppurations may develop in two ways: (1) by direct propagation and (2) by glandular inflammation and suppuration and diffusion through the lymphatic vessels. The parapharyngeal suppurations can be divided into two main groups: (*a*) anterior and (*b*) posterior. In group *a* two types can be distinguished, (1) the anterior superior abscess, or the pterygoid-pharyngeal abscess, and (2) the anterior inferior abscess passing down to the submaxillary and submental region. In group *b* can be distinguished (1) the posterior-superior parapharyngeal abscess, which passes medially to the styloid group of muscles, appears in the suboccipital region and from there passes down in the posterior triangle of the neck, originating from either direct propagation or glandular suppuration, and (2) posterior-inferior parapharyngeal adenophlegmon, which passes down along the sheath of the vessels of the neck, originating only from the lymphatic glands along the jugular vein. Although these four types can be separated distinctly, transition forms can be found. Probably an abscess from the pterygoid-pharyngeal space can pass downward and sometimes does. It must be remembered that the distances are rather small. It is self evident that the virulence of the bacteria and the individual resistance are of great importance for the course of the inflammation. The author gives a description of each of the types mentioned and outlines the methods of surgical treatment.

EDITOR'S ABSTRACT.

HISTOLOGIC STUDIES ON TONSILS AND ADENOID GROWTHS. E. URBANTSCHITSCH, Monatschr. f. Ohrenh. **70**:1177 (Oct.) 1936.

Urbantschitsch describes the histologic aspects of the tonsils and adenoids removed from 446 patients. All tonsils and adenoids that were histologically examined showed chronic inflammatory changes. In addition, calcareous deposits were discovered 7 times and fungus colonies 33 times. In 25 cases cartilaginous or bony tissues were observed in the tonsillar bed. In 5 cases epithelioid cell tubercles were observed and subsequent roentgen examination revealed tuberculous processes in the thoracic cavity. Thus the histologic examination of the tonsils and adenoids brought about the diagnosis and early treatment of the tuberculosis. In 2 cases a carcinoma was detected in the tonsils.

EDITOR'S ABSTRACT.

TONSILLECTOMY BY SHORT WAVE. L. H. STIEBÖCK, Wien. klin. Wchnschr. **50**: 1039 (July 9) 1937.

Stieböck explains that the apparatus used in the treatment in question has an efficiency of 1.5 kilowatts. This gives continuous vibrations up to 50,000,000 a second, and the rays are from 4 to 8 meters in length, depending on the frequency.

A newer 200 watt "superfrequency" unit can produce from 500,000,000 to 1,000,000,000 oscillations per second and rays with a length of from 80 to as low as 32 cm., with a comparative increase in therapeutic value.

Diathermy, on the other hand, produces rays with a length of 300 meters and a frequency of only 1,000,000 per second. In diathermy and other bipolar methods the patient is included in the circuit, but in the short wave treatment, which is unipolar, the energy is applied only to a given area. This rules out the possibility of uncontrolled flow of current and consequent shock.

In tonsillectomy, as well as in the treatment of papillomas, condylomas and other growths by the means described, the short waves exert a beneficial caloric effect, although this warmth, it should be expressly noted, is to be studiously avoided under other circumstances.

Stieböck hopes to be able with the superfrequency treatment to relieve stenocardiac complaints. In collaboration with O. Förster, Vienna, he perfected another invention, which will release positive ions and ground them by way of affected tissues. This "anode," or "positive pole," therapy has been used successfully in the treatment of various patients with eczema and involvement of the joints.

A. F. ABT, Chicago. [AM. J. DIS. CHILD.]

Larynx

INFLAMMATORY TUMORS OF THE TRUE VOCAL CORDS: DIRECT LARYNGOSCOPIC OBSERVATIONS. G. TUCKER, J. Laryng. & Otol. **51**:563 (Sept.) 1936.

Tucker presents his observations on the diagnosis and treatment in a series of cases of inflammatory tumor of the larynx and demonstrates a direct laryngoscopic method of treatment, with its results. Regarding the frequency of occurrence, he says that in a recent five year period he observed in his practice and at the Bronchoscopic Clinics of the University of Pennsylvania 145 cases of chronic inflammatory tumor of the true vocal cords. Thus the occurrence of this condition is more frequent than is indicated by the cases reported in the literature. He discusses the pathology, etiology, symptomatology, diagnosis, prognosis and, finally, treatment. Regarding treatment, he says that when the tumor is small, absolute vocal rest, with the patient using a pencil and paper for all communication, may be tried. When the lesion on the true cord is localized, shaving off the mass of tissue will restore the normal form of the cord. A careful technic is required so that the deeper structure of the cord is not injured. Direct examination with the anterior commissure laryngoscope and the cupped forceps of Chevalier Jackson is the method of choice. The individual operator should

use the method in which he is most skilled and by which he can obtain the best results. After describing a number of illustrative cases and presenting sketches of the larynx before and after direct laryngoscopic treatment, the author reaches the following conclusions: 1. Chronic inflammatory tumors of the true vocal cords tend to increase in size because of the functional activities of the cords. 2. A vicious circle is set up, in which local irritation, increase in the size of the tumor and overactivity of the musculature of the larynx are important etiologic factors. 3. Restoration of the normal contour of the cord by direct laryngoscopic removal of the tumors and by voice training to restore the normal muscular action will eliminate the vicious circle and restore the larynx to normal in most cases. The microscopic examination of the tissue obtained by direct laryngoscopic removal provides a histologic basis for diagnosis.

EDITOR'S ABSTRACT.

SURGICAL THERAPY OF CHRONIC STENOSES OF THE LARYNX. A. ŠERCER, Monatschr. f. Ohrenh. **70**:1153 (Oct.) 1936.

Šercer says that there is no general rule for the treatment of all laryngeal stenoses and that in a number of cases a cicatrized tracheostomy without a cannula is all that can be accomplished. However, in reviewing his material on laryngeal stenosis, he observed a particular type of stenosis which is characterized by permanent median fixation of the vocal cords. The median stenoses develop either as the result of bilateral paralysis of the posterior cricoarytenoid muscle or as the result of median fixation of the cricoarytenoid joints. Since the condition of the larynx is always the same, the author reasons that the same treatment ought to be effective. At first he employed submucous chordectomy, but this always had to be followed by prolonged dilation. However, in the last 5 cases he employed a method which he designates a submucous exenteration of the larynx. In this process of laryngeal exenteration, not only the vocal cords are removed but that portion of the arytenoid cartilage which protrudes beyond the internal rim of the cricoid cartilage into the laryngotracheal tube. The vertical portion of the arytenoid cartilage, however, should be preserved, because it belongs to the supporting framework of the larynx. The operation is performed in two stages. During the first, laryngofissure and exenteration are done, and after the wound has healed the laryngostoma is left open and without a cannula for several days. Then the stoma is covered with a plaster, and the patient is carefully observed. After it has been determined that the breathing is adequate, the second stage of the operation, the plastic closure of the laryngostoma is done.

EDITOR'S ABSTRACT.

PHYSIOLOGIC AND HISTOLOGIC ANALYSIS OF THE VAGUS AND OF THE INNERVATION OF THE LARYNX. A. RÉTHI, Monatschr. f. Ohrenh. **70**:1159 (Oct.) 1936.

Réthi points out that in his earlier series of reports on the innervation of the laryngeal nerves he proved that the laryngeal muscles have a double innervation. The experiments described in that series were made on the larynx, but the author thinks that, in order to gain a better understanding, it is advisable to study the origin of the laryngeal nerves, that is, the vagus and the cranial nerves connected with the larynx, viz., the glossopharyngeus, the accessorius and the hypoglossus. In its intracranial portion the vagus is entirely free from foreign fibers, but after it passes through the jugular foramen it receives anastomoses from the aforementioned nerves. The author describes several experiments on dogs. He found that severing the accessorius results in paralysis of the homolateral vocal cord during respiration but not during the process of swallowing. The cutting of the recurrent nerve on one side in another dog had the same effect; that is, the homolateral vocal cord was paralyzed during respiration and phonation but not during the act of swallowing. After the severing of the accessorius nerve, the muscular branch of the recurrent (inferior laryngeal) nerve degenerates, whereas the superior laryngeal nerve and its branches show no signs of degeneration. When in another dog the recurrent nerve was severed, its muscular branches, which

were distal to the site of cutting, degenerated, whereas the anastomosis between the superior and the inferior laryngeal nerve and the tracheal branches of the recurrent nerve, which contain the fibers of that anastomosis, remain unimpaired.

EDITOR'S ABSTRACT.

Miscellaneous

ALLERGY IN CHILDREN. H. J. RINKEL, Arch. Pediat. **54**:349 (June) 1937.

An analysis of the findings in 25 children with asthma, 25 with hay fever and 25 with perennial nasal allergy is compared with an analysis of the findings in similar numbers of adults with the same diseases. The comparison fails to support the assumption that food is a more common cause of asthma in the child than in adults.

ORR, Buffalo, N. Y. [AM. J. DIS. CHILD.]

ROENTGEN RAYS IN THE TREATMENT OF ACUTE CERVICAL ADENITIS. S. HURWITZ and S. N. ZUKERMAN, J. Pediat. **10**:772 (June) 1937.

Sixty-two infants and children suffering from acute cervical adenitis were treated with roentgen rays. Twenty-one of these patients were hospitalized, and 41 were treated in the outpatient clinic. The dose used on the patients in the hospital was from 10 to 20 per cent of the skin erythema dose, repeated every three to five days. Treatment was begun from two to twenty-eight days after the onset, the average time being eight and three-tenths days. The number of treatments ranged from one to five. Resolution occurred in 17 (80.9 per cent) of the patients treated with roentgen rays as opposed to 10 (58.8 per cent) in a control group. Suppuration resulted in 3 (14.3 per cent) of the treated patients, while 7 (41.2 per cent) of the control group showed abscess formation. Among the children treated in the outpatient clinic, on whom a dose of from 70 to 200 roentgens was used, from one to three treatments, at intervals of from two to seven days, was found sufficient to bring about resolution in 35 (85.4 per cent). Suppuration occurred in 6 patients (14.6 per cent). The striking features of this method of treatment were rapid subjective improvement and accelerated termination. Radiation was most beneficial in the early stages of inflammation, absorption being the usual final result. In cases in which suppuration had already occurred, radiation hastened necrosis and early centralization of infection, thus minimizing surgical intervention and enhancing the cosmetic effect.

RAUH, Cincinnati. [AM. J. DIS. CHILD.]

CEREBRAL SINUS THROMBOSIS IN CHILDREN. J. H. EBBS, Arch. Dis. Childhood **12**:133 (June) 1937.

Thrombosis of the cranial sinus, although comparatively rare, appears to be increasing. Ebbs gives summaries of 8 cases, with important facts relating to 32 cases recorded during fourteen years. A review of the principal signs and symptoms in these cases suggests that there is a fairly constant clinical syndrome in the majority of those cases in which the condition is not due to mastoiditis and cellulitis with involvement of the lateral and cavernous sinuses, respectively. In this group the following points often assist in making a correct clinical diagnosis of sinus thrombosis: sudden onset of restlessness followed by convulsions; localization of the convulsions as shown by twitching, followed by paresis; cerebrospinal fluid findings of increased or normal pressure, normal or slightly increased number of cells, the presence of red blood cells or a xanthochromic character of the fluid, sterile cultures, low sugar content, high content of chlorides and normal or slightly raised level of protein. The other points in the condition are the drowsiness or semiconsciousness and the absence of evident infection during the course of the illness. Infection is practically always observed at postmortem examination. The high level of chlorides in the cerebrospinal fluid is emphasized as an aid in arriving at the diagnosis. Several attempted explanations of the thrombosis are reviewed, but no reference is made to treatment or to pathologic details.

KELLY, Milwaukee. [AM. J. DIS. CHILD.]

Society Transactions

MASSACHUSETTS EYE AND EAR INFIRMARY

Regular Weekly Clinical Meetings from August 1936 to April 1937

EDITED BY R. L. GOODALE, M.D., BOSTON

FOREIGN BODY IN THE ESOPHAGUS. DR. H. G. TOBEY, DR. P. MYSEL and DR. H. D. SMITH.

This is the case of a 2 month old infant who swallowed a small safety pin one week before his entry to this hospital. He was rushed to the hospital at Brattleboro, Vt. There he seemed to be having a little difficulty in swallowing but no other symptoms. He was sent here, where roentgenograms showed a small pin in the upper third of the esophagus. It was a small pin; the point was to the left and had not penetrated the wall. The day after the patient's entry the esophagoscope was used by Dr. Mysel.

DISCUSSION

DR. P. MYSEL: This baby had swallowed the pin one week before I saw him. It seemed to me that it was wise not to attempt to close the pin and extract it by way of the mouth. So under fluoroscopic control the esophagoscope was used. The keeper of the pin was readily seen, about 2 or 3 inches (5 to 7.5 cm.) below the mouth of the esophagus; under fluoroscopic control it was simply pushed along the esophagus into the stomach. The patient did very well; within twenty-four hours he passed the pin, and within forty-eight hours after that it was possible to send him home. He made an uneventful recovery. He had no postoperative reaction. This patient was put under avertin (tribromethanol) anesthesia, about 80 mg. of avertin being used. I think this is one of the first cases I have observed of an open safety pin in the esophagus of a 2 month old child.

DR. H. G. TOBEY: With a 2 month old baby the difficulty is that a safety pin closer which will enter the esophagus may not be large enough to hold the keeper of the pin. If not, it is necessary to push the pin into the stomach. I am glad to see that the fluoroscope was used. There is some danger in pushing the pin down; often it may rotate in the forceps and possibly perforate the esophagus.

FOREIGN BODY OF THE ESOPHAGUS. DR. H. G. TOBEY, DR. P. MYSEL and DR. H. D. SMITH.

This is the case of a baby 16 months old who at about 4 o'clock the day before entering the hospital, while he was being undressed, seized a safety pin and swallowed it. He vomited a number of times and spit up a small amount of blood. He was not given anything by mouth but was brought here, where roentgenograms were taken.

DISCUSSION

DR. A. S. MACMILLAN: The roentgenograms show the pin to be sticking through the mouth of the esophagus, the guard being in the pharynx itself. The pin is about half in the pharynx and half in the esophagus. It is transverse and on a level with the seventh cervical vertebra.

DR. P. MYSEL: Dr. MacMillan felt that this pin had perforated the esophagus. The child had a normal temperature. I believe there had been no previous attempt to remove the pin. With the patient under avertin anesthesia and with the aid of Dr. Mosher's speculum, the pin was seized just below the keeper. By rotating

it, it was possible to pick out the pin without any trouble. There was no bleeding after this extraction.

Dr. H. D. Smith: The next day the patient had a little fever. He swallowed easily. The following day his temperature was slightly higher. He had a little tenderness along the left side of his neck and a little induration along the left sternocleidomastoid muscle. His temperature is coming down today. There seems to be no increase in the swelling, and the child looks much better than he did after the extraction. Of course, the swelling may break down and form a localized abscess at the point of the perforation. In any event, it will be possible to reach the infection if it does suppurate through an external incision. At the present time it looks as though the cellulitis might quiet down without forming an abscess.

Dr. A. S. MacMillan: A roentgenogram taken the day after removal of the pin showed a small amount of air at a point where one would expect a perforation to be. A swelling arose in the upper end of the esophagus and extended into the pharynx. It did not extend to the mediastinum.

This morning there is less swelling in the pharynx; what there is is in the midline behind the trachea, pushing it forward considerably and narrowing it somewhat. There is no more air.

Dr. P. Mysel: The child has a nasal feeding tube in now to keep him from swallowing any more than is absolutely necessray. The swelling may clear up, and it may break down and possibly have to be drained from the outside.

Foreign Body in the Bronchus. Dr. L. A. Schall and Dr. L. H. Heine.

A child 2½ years of age was admitted to the infirmary with a history of having swallowed a fairly large safety pin. Roentgenograms were taken, which showed the safety pin to be in the right main bronchus.

DISCUSSION

Dr. A. S. MacMillan: In this roentgenogram the pin is closed and the point is down. This is the first case reported here of a safety pin closed and point down.

Dr. L. A. Schall: The extraction was done with ether anesthesia. About a fourth of the pin was projecting into the trachea. It was in the main stem bronchus and was too large to go farther down. With the grasping forceps and the aid of the fluoroscope the pin was seized and easily delivered into the larynx. The patient has had no complications. This is the first closed safety pin I have ever seen in the bronchus.

Mucocele of the Frontal Sinus. Dr. L. A. Schall, Dr. R. L. Goodale and Dr. L. H. Heine.

A 55 year old Italian woman entered the hospital thirteen days ago. She had noticed a swelling above her right eye for nine months. The swelling had evidently increased little in size since it was first noticed. She had no other symptom: no pain, no headache and no disturbance of vision.

Examination showed numerous polyps in both nares and considerable pus in the region of both middle meatuses. The right palpebral fissure was more narrow than the left, and there were a supraorbital swelling and fulness, which measured 2.5 by 2.5 cm., over the inner angle of the frontal bone. This fulness was tense and had the feel of a rubber ball. It was translucent. There was slight limitation of movement of the eye in all directions.

DISCUSSION

Dr. A. S. MacMillan: The roentgenograms showed pansinusitis on the left side and a very clear frontal sinus on the other. The frontal sinus is altogether too clear and the outline was smooth. It is not very often that one sees a sinus without any partitions projecting at all.

DR. L. H. HEINE: Eleven days ago Dr. Goodale did an obliterative operation on the frontal sinus.

DR. R. L. GOODALE: When I started I had in mind doing an external ethmoid and Lynch operation, but on exposing the frontal bone I found that most of the frontal sinus and a good bit of the supraorbital ridge and of the anterior wall were gone. On opening into the frontal sinus and taking out the membrane along the floor, I found in the posterior wall a large defect through which there was a herniation of dura. This looked liked a mucocele at first sight. Therefore, I changed my plan and took off the entire anterior wall and cleaned out all the ethmoid cells. The membrane from the frontal sinus stripped off very easily except where the dura was exposed. Here it was adherent.

DR. H. P. MOSHER: How much of the posterior wall is gone?

DR. R. L. GOODALE: I should say that at least three fourths of the posterior wall is gone.

DR. H. P. MOSHER: It looks as though nature tried to do an obliterative operation on the frontal sinus—as though she were trying to give a hint that one should take off the front wall and the posterior wall and do as little as possible when one tries to obliterate a frontal sinus. The thing that bothered me was that Dr. Goodale said that the dura was very adherent. It may be that it is entirely adherent and cannot be taken off. It may be that enough dura is exposed and that this will produce obliteration. If not, there is the question of taking off more of the bone.

DR. R. L. GOODALE: How much does one have to worry about the mucous membrane over the dura?

DR. H. P. MOSHER: I think the granulation will take care of that. In this case granulation is proceeding nicely.

Is it not true that in cases of osteomyelitis there have been more successful obliterations after removal of the anterior and the posterior wall than after any other procedure? I think the day will come when that will be the operation of choice for obliteration.

DR. H. P. CAHILL: How about doing a Lynch operation to empty the mucocele? One can always get a very large opening through the nose in cases of mucocele. Dr. D. C. Smyth did that repeatedly, and I never knew him to have any bad results.

DR. G. L. TOBEY: I think that with the dura exposed it is better to obliterate the sinus than to stand the chance of infection afterward.

DR. H. P. CAHILL: I would rather have obliteration effected through the nose.

DR. C. T. PORTER: Unless one takes all the frontal wall off, one will leave places in the outer angle that will become infected.

DR. G. L. TOBEY: The situation is exactly the same as in a case of osteomyelitis.

DR. G. B. FRED: In the case reported the dura was so far forward there would not have remained a good opening into the nose even though obliteration had been done. I do not know why.

DR. H. P. CAHILL: A yielding dura like that arouses suspicion.

DR. G. B. FRED: It was almost cystic.

MIXED TUMOR OF THE TONGUE. DR. H. G. TOBEY and DR. H. D. SMITH.

This is the case of a 46 year old man who seven weeks ago began having a sensation of a lump in his throat and excessive salivation. This condition became slowly worse. He had no other symptoms, such as pain radiating upward or difficulty in swallowing. On examination, a red, circular, mulberry-like, hard mass about 1.5 cm. in diameter was found in the area of the left lingual tonsil. It seemed to run down nearly to the base of the epiglottis. About four days after the patient entered the hospital, the tumor was removed by Dr. Tobey and Dr. Garland.

DISCUSSION

DR. H. G. TOBEY: This tumor seemed to be in a very favorable position for the use of Dr. F. E. Garland's method of removal. This method consists of the following procedures: The tongue is gradually extended to one side by means of traction applied through successive sutures placed in the side of the organ. On full extension of the tongue, the base of the anterior pillar is found to be in easy reach. A suture is placed through this tissue, some of the mucous membrane of the floor of the mouth being included. The tongue is then incised between these last two sutures and the dissection carried on by means of scissors. As the dissection proceeds, more sutures may be placed through the incised portion of the tongue, bleeding being thereby controlled and further traction and extension allowed. By this means the entire area of the floor of the mouth down to the base of the tongue, including the lateral wall of the pharynx, is made easily accessible in a bloodless field. After the removal of the tumor, the floor of the mouth is sutured and all traction sutures which are in relation to the incision are tied, all other sutures being removed. This procedure gave an excellent approach to the described tumor and allowed for its easy dissection. After this type of operation there is usually some edema of the tissues; this, however, has never been of serious extent.

The patient we operated on seems to be doing well now. I think an epiglottis can easily be taken off in this way by approaching from the side. It is somewhat analogous to exposing the larynx with the laryngoscope on the side of the tongue rather than from the center.

DR. H. D. SMITH: The day after the operation the patient was comfortable, but he had slight edema of the tongue and of the upper part of the left side of the neck; this seems to be going down at the present time. He has had no respiratory difficulty at any time.

DR. H. G. TOBEY: The operation was done (with general anesthesia) with the patient sitting up. I think it could have been done perfectly well if he had been lying down.

THROMBOSIS OF THE CAVERNOUS SINUS WITH ABSCESS OF THE BRAIN. DR. G. H. POIRIER and DR. H. D. SMITH.

A 42 year old man came into the hospital with a history that ten days previously he had contracted a grippy infection; about two days before his entry this seemed to localize in the right frontal area of his head. The day before entry he showed ptosis of the right upper eyelid and diplopia, and the visiting family doctor felt that he had papilledema of both eyes. He was sent here. He seemed sick. His white blood cell count was 9,000.

He had headache so severe that administration of 1 grain (0.06 Gm.) of codeine was necessary to keep it under control. He had complete ptosis of the right eye, a little exophthalmos and restriction of motion of the right eye in all directions. Physicians of the ophthalmologic service saw him and were unable to find any papilledema in either eye. He was seen by members of the neurologic service. They could not find anything except the ocular symptoms, but the impression of the house officer was that there was an abscess of the right frontal lobe of the brain, with pus posterior to the right orbit. A lumbar puncture was done the day after the patient's entry, and the fluid was found to be entirely normal. There were normal initial pressure and 4 cells, and the colloidal gold reaction was entirely normal.

Four days after his entry, the headache still persisted. The right eye showed a little motion. The edema of the lid went down. During the fourth night he complained of severe pain, and in the morning he showed complete ptosis of the left eye. He had inflammation and tenderness of the lids, chiefly of the left eye, and headache in the frontal area. The right eye seemed improved. The severe headache has continued; the right eye has improved slowly. The left eye has remained about the same.

One week after his entry, for the first time the patient had a little papilledema in the right eye, slightly less than 1 diopter. The left eye had a trifle more than the right eye. Also for the first time, he showed slight stiffness of the neck and a slight Kernig sign. He seemed much worse that day and complained of a great deal more headache and of a rather peculiar weakness of the left side. He had a severe chill about ten days after his entry. After the chill he had paresis on the left side during which he had a generalized convulsion, which lasted for one and one-half minutes. He had repeated blood cultures, four in all, and all were reported negative. He was seen again by Dr. Ayer, of the neurologic service. A lumbar puncture was done, and this time the initial pressure was 450 and there were 13 cells. There were a slight rise in the colloidal gold reaction and an increase in the sugar content of the spinal fluid. At that time it was felt that he probably had bilateral thrombosis of the cavernous sinus.

The next day he again had chills. A third lumbar puncture was done, because the stiff neck seemed to be more pronounced. This time the initial pressure was 350. The cell count was still low (15) and the sugar was still present, but no other abnormality was found in the fluid. He gradually became worse and had an average of three or four generalized convulsions each day. Two days after the last lumbar puncture was done he died.

He had had repeated roentgen examinations. They did not show evidence of any pathologic condition in the sinuses, the base of the skull or the mastoid or the petrous bone. There was questionable thickening in the left lower lobe of the lungs, which apparently cleared up in a day or so. The only foci we could find outside of the grip were some very bad teeth.

At autopsy it was observed that both sphenoid sinuses were full of pus and that the pus had ruptured through the roof, so that it surrounded the sella turcica. The entire right frontal lobe of the brain was essentially one large abscess. There were a retrobulbar abscess on the left side and an abscess of each cavernous sinus, with pus. The only other abnormal condition was chronic adherent peritonitis.

DISCUSSION

DR. G. H. POIRIER: This is a very interesting case. For the first week or ten days the signs and symptoms pointed to thrombosis of the cavernous sinus, but the autopsy showed very definite acute abscess of the frontal lobe, most likely secondary to the acute nasal infection.

DR. C. T. PORTER: Was the paresis due to the extension of the abscess in the motor area? This is very frequently the case with an abscess of the frontal lobe which causes pressure on the motor area and the internal capsule.

DR. G. H. POIRIER: The area of the acute abscess of the brain was the size of the hand.

DR. S. CLINE: Was there any clinical evidence of sphenoiditis?

DR. G. H. POIRIER: No. Roentgenograms were taken one week or ten days before autopsy, and they showed only some thickening of the sphenoid.

DR. H. G. TOBEY: Probably the focus was the sphenoid. The only way to prove anything was to open the sphenoid. But the patient was too sick to stand an operation.

OSTEOMYELITIS OF THE FRONTAL BONES. DR. H. P. CAHILL, DR. F. L. WEILLE and DR. L. A. BROWN.

This patient, 19 years of age, came into the hospital on Sept. 14, 1936. Her history dates back to 1929, when she came to the clinic with nasal obstruction on the left side. At that time polyps were removed from the left naris. She had them removed again four times in the next two months. This procedure was followed by four irrigations of the left antrum and finally by a Caldwell-Luc operation. She came in again in 1932 with nasal obstruction on the left side

due to polyps and pus in the left naris. This time she had an external operation on the ethmoid and revision of the Caldwell-Luc operation. One month after discharge she returned, with osteomyelitis of the front wall of the left antrum. No further operating was done, but sequestrums were removed during her stay in the hospital. She was free from symptoms for three years, until in 1935, in a motorcycle accident, she received a deep gash over the bridge of her nose. After this accident she noticed nasal obstruction on the right side for the first time. She came in again in July 1936, with complete blockage of the right naris due to pus and polyps. An external ethmoid operation was done on the right side. This was followed in a few days by a radical antral operation on the right side. She went home in the middle of August and came back on September 14, with swelling over the bridge of the nose. At that time the floor of the frontal sinus was removed. In the frontal sinus there were polyps and pus.

Ten days later there were a little edema of the forehead and more swelling over the scar on the left side from the operation of three years previously. This was incised, and there was escape of pus. On October 3 roentgenograms were made, since the edema did not go down.

DISCUSSION

DR. A. S. MACMILLAN: When she came in in July, the roentgenogram showed the pansinusitis on the right side for which she was operated on. The next film showed the operative defect on the left side and on the right side. This film was taken after the operation. It showed also the moth-eaten appearance of the front wall of the frontal sinus above the operative defect. The lateral view taken at the same time showed better the amount of destruction taking place in the front wall of both frontal sinuses.

DR. L. A. BROWN: On October 1 a diagnosis of osteomyelitis of the frontal bone was made. On October 3 the patient was operated on by Dr. Weille.

DR. F. L. WEILLE: This girl had edema in the midline within a fingerbreadth of the hair line and laterally almost to the temporal fossa while the roentgenogram showed destruction of the front wall of the frontal sinus only. The incision was made with a knife. I used the principle of Dr. Cushing of putting snaps on the periosteum and letting the weight of the snaps control the bleeding. Drill holes were made in good bone from 1 to 1½ inches (2.5 to 3.8 cm.) beyond the diseased area. The zygomatic process was extremely heavy; it was hard to get off the bone laterally, even though the medial portion of it was diseased. The plate of frontal bone was removed. The floor of the frontal sinus was removed widely from the right side. Some orbital fat was exposed on the left side. The patient was in good condition at the end of the operation, and this morning she is starting to get up.

DR. L. A. SCHALL: Was there any pus in the bone?

DR. F. L. WEILLE: The bone was grossly diseased over the anterior wall; there was no pus in the diploe. There were no extradural abscesses.

DR. H. P. MOSHER: This seems to have been done according to Hoyle; pulling back the periosteum after Dr. Cushing's fashion has been done off and on here. It should be done more often, because it controls bleeding. The big point is that the osteomyelitis can run around to the side. One should be suspicious of osteomyelitis way around in the temporal fossa in all of these cases. I am very much pleased with the size of the specimen, although it does not go to the hair line.

DR. A. S. MACMILLAN: The postoperative roentgenogram shows the upper limit of the frontal bone but does not show osteomyelitis beyond it. The anterior wall at this point of the frontal sinus is gone. The bone which is seen seems to be healthy in the front wall.

BEZOLD'S ABSCESS. DR. P. E. MELTZER, DR. M. H. LURIE and DR. H. D. SMITH.

This is the case of a man aged 21 who six weeks ago had a cold and pain in the left ear. He had a paracentesis done, and the ear discharged for six weeks. He did not return to the outpatient department.

He had no other symptoms until, one day before entry, while riding in a car, he noticed that his left ear was standing out and that it was painful. When he entered the hospital the next day, he showed marked postaural tenderness, with a brawny induration extending below the tip of the mastoid. The drum was thick and had a central perforation.

The next day he was operated on by Dr. Meltzer and Dr. Smith. A simple mastoidectomy was done on the left side. There was a perforation of the cortex near the tip about 1 cm. in diameter. Pus welled up out of this perforation. The cells throughout the mastoid were found to be markedly broken down, and there was frank pus down in the tip and up around the antral region. The sinus was covered by a large perisinal abscess with dirty granulations, which were cleaned off. The sinus was thought to be thrombosed. Heat was applied to the man's neck, and he got along very well. There was a perforation through the cortex just above the tip but not at the bottom. The tip was taken off. Culture of material from it showed pneumococci.

DISCUSSION

DR. H. P. MOSHER: There have not been any occurrences of Bezold's abscess for some time. Now it is coming back again.

BEZOLD'S ABSCESS. DR. P. E. MELTZER, DR. M. H. LURIE and DR. H. D. SMITH.

The next case is that of a man aged 52 who four weeks before entry to the hospital had a severe cold. Three weeks before entry he had spontaneous discharge from the right ear. One week before admission he had three attacks of a chilly sensation. He had some headache in the right temporal region. Three weeks before entry he had considerable pain in the right ear, with postaural swelling and tenderness over the tip of the mastoid. It extended down the neck to a point about halfway between the angle of the jaw and the clavicle. He came in with a marked brawny swelling, about 2 inches (5 cm.) down from the tip. It did not seem to extend more than halfway up the mastoid bone. The drum was beefy red, and there was mucopurulent discharge from the middle ear.

DISCUSSION

DR. A. S. MACMILLAN: The roentgenogram shows a very small left mastoid, with no cells in it. There is no gross destruction at the tip; it is perfectly solid. In contrast with this, the roentgenogram in the first case showed a very large mastoid with complete destruction of the tip.

DR. H. D. SMITH: Six days ago a mastoid operation was done by Dr. Lurie and me. On removal of the cortex, which was thick, pus flowed under pressure from both the tip and the area around the antrum. There was a definite perisinal abscess in this case also. The sinus was exposed. Apparently there was no thrombosis of the sinus. There were found to be pus and granulations extending deep under the facial ridge in a large mass of retrofacial cells; it was felt that this condition was probably a lead. Since the operation, the postaural wound has drained continuously and the swelling has subsided. In this case, as in the first and third cases, pneumococci were found.

DR. M. H. LURIE: This case was interesting in that the perisinal abscess was very low. On exposing the retrofacial region, we found that the infection had not really perforated through the mastoid but was following down along the sinus, in the region of the jugular bulb and so on down into the neck. With poulticing linseed the tremendous cellulitis and the large glands in the neck have gradually been reduced.

BEZOLD'S ABSCESS. DR. P. E. MELTZER, DR. M. H. LURIE and DR. H. D. SMITH.

The third case is that of a man aged 47 who eight weeks before entry had a cold. One week later he had two paracenteses in the left ear. The ear discharged for about two weeks and then apparently cleared up. As the patient was deaf, the ear was blown out. Afterward he had pain. The pain lasted one week and gradually cleared up. One week later pain again developed about the left ear, and it again discharged for two days. With this pain he had a little swelling in the neck, which gradually increased until he arrived at the hospital, four weeks later. He then showed an apparently normal drum, no perforation and no discharge. All landmarks were present. There was a large brawny swelling extending from the tip of the left mastoid two thirds of the way down toward the clavicle. There was marked postaural tenderness over the tip and about halfway up over the mastoid process.

A simple mastoid operation was done on the left side by Dr. Lurie. The antrum was sclerosed. Pus was in the tip; it was completely destroyed. A lead was found deep to the tip, which apparently extended to the neck. Ten days after operation the mass in the neck became fluctuant. It was opened and a large amount of pus obtained. The wound in the neck began to close about five days ago.

DISCUSSION

DR. M. H. LURIE: These 3 cases of Bezold's abscess show three of the possible ways in which the infection can travel in the neck. In the first case there was a posterior perforation through the posterior portion of the digastric groove. The infection extended to the tip, becoming localized. In the second case the infection followed along the lateral sinus and the jugular bulb and presented itself along the region of the sternocleidomastoid muscle and then tended to follow posteriorly down the fascia of the deep vessels of the neck. In the last case mentioned the infection had evidently drained itself through the tip and into the subcutaneous tissues of the neck, and as a result of the draining of this mastoid infection the ear tended to clear up. With the walling off of the abscess in the neck there was a return of symptoms in the ear, and the draining of the infection in the neck, with the mastoid operation, was all that was necessary.

CHRONIC SUPPURATIVE HEMORRHAGIC OTITIS MEDIA ON RIGHT SIDE—CHRONIC DESTRUCTIVE LABYRINTHITIS, FACIAL PARALYSIS AND MENINGITIS—WITH LABYRINTHECTOMY. DR. H. G. TOBEY, DR. P. MYSEL and DR. L. H. HEINE.

A man aged 30 came into the hospital on Dec. 4, 1936, complaining of a severe frontal headache. He was nauseated and had vomited for a few days before his entrance. He had facial paralysis and a stiff neck, and Kernig's sign was present on both sides. Lumbar puncture was done. The initial pressure of the spinal fluid was 600; there were 5,600 cells, of which 84 per cent were polymorphonuclears. The total protein content was high, the Pandy reaction was positive, sugar was present and there was a question of organisms' being present in the fluid on direct smear. The patient had had severe pain in the right ear for the two weeks before entry. This ear, which had been discharging intermittently for twenty-eight years, had ceased to discharge at the onset of the pain. His vestibular and acoustic functions were completely gone. The caloric test with cold water showed no reaction. He was too sick to be turned. He was stone deaf in the good ear according to the noise apparatus. The fundi showed some papilledema. Roentgenograms showed destruction about the antrum and sclerosis of the mastoid. The patient had no spontaneous nystagmus. The condition was evidently chronic destructive labyrinthitis with beginning meningitis.

On examination of the ear, there were no postaural signs but there was a large marginal defect with some foul secretion in the external canal.

DISCUSSION

DR. A. S. MACMILLAN: In the roentgenograms the left mastoid is normal, very large and pneumatic; the right mastoid is completely sclerosed. The antrum is enlarged, but not very much. It is certainly not a cholesteatomatous enlargement. The petrous tips appear normal.

DR. L. H. HEINE: On entry this patient had a temperature of about 102 F. and his pulse was elevated; outside of that his chart showed nothing remarkable.

DR. P. MYSEL: I think one of the favorable features in this case was that with the obvious clinical findings of meningitis the patient's temperature was not elevated to any great extent. He was operated on the night of the day he entered the hospital; a hand drill was used to enter the antrum. As it entered there was a gush of straw-colored fluid, not pus. No culture was taken of this fluid. The hand drill has its advantages and disadvantages. The advantage for this type of operation is that it does not shake the patient's head, as a mallet and chisel do. The disadvantage is that it handicaps the operator in that he does not get the desired amount of room; he is working, of course, in a more or less deep cavity all the time. The cortex was very thick and very difficult to remove. As the region of the antrum was approached, a mass of granulations was found between what was the horizontal canal and the posterior canal. When the granulations were removed, a fistula was seen in the horizontal canal and in the posterior canal. The promontory was removed, and both the round and the oval window were found. A probe could be passed from the posterior part of the vestibule into the middle ear. The facial nerve was lying in a sequestrum that was part of the anterior wall of the external canal. Every time this sequestrum was disturbed the face twitched.

The next day the headache was much less pronounced. Lumbar puncture showed a decrease in initial pressure of the spinal fluid from 600 to 300. On the third day the patient was very much more improved. His cell count dropped to 100. Lumbar punctures were not repeated, because the cell count rapidly diminished and the symptoms cleared up. Cultures of the spinal fluid did not yield any organisms. The facial paralysis cleared somewhat after the operation. When the patient came out of the anesthesia he was able to close his eye better than he does at present, but the next day there was a definite increase in the facial paralysis. The electrical reactions show that there is no response to the faradic current and that stimulation with the cathode is greater than with the anode. It is too soon to give the prognosis; it may take a week or two. The question now is what to do about the facial nerve. The sequestrum is still attached to the nerve.

DR. H. P. MOSHER: One should decompress it.

DR. H. G. TOBEY: I feel that as soon as signs of meningitis are gone, the sequestrum must be removed; otherwise it is going to keep on causing suppuration indefinitely until it comes out of itself. I think in removal of the sequestrum a certain amount of decompression of the nerve will be done. If any swelling of the nerve is present, one can simply open the sheath.

DR. H. P. MOSHER: I agree to that.

Dr. Wherry, how do you feel about using the hand gouge rather than the ordinary instruments in such operations as this?

DR. W. P. WHERRY: My associates and I are using the gouge now. We use the Hudson drill. If the points become dull, we have to resort to the gouge again. We have an electrical machine that works most of the time. We carry the Hudson drill to use in an emergency, when the gouge is not working.

DR. P. MYSEL: One is certainly handicapped with a Hudson drill, because it gives too much depth and one is working in a deep cavity all the time.

DR. C. T. PORTER: I agree with Dr. Tobey about taking care of the facial nerve. One should be sure the patient has recovered from the meningeal signs

and then get the sequestrum out. I do not like the drill in the mastoid. It fills up all the space. One is looking into a small hole.

DR. H. P. MOSHER: I feel very much that way but hate to act contrary to Dr. Cahill's advice.

CHRONIC SUPPURATIVE HEMORRHAGIC OTITIS MEDIA ON LEFT SIDE—ACUTE DESTRUCTIVE LABYRINTHITIS, FACIAL PARALYSIS AND MENINGITIS—WITH LABYRINTHECTOMY. DR. H. G. TOBEY, DR. P. MYSEL and DR. L. H. HEINE.

This man, 49 years of age, has had chronic aural disease for forty-five years. He came into the hospital complaining of severe headache in the temporal region on the left side, nausea and vomiting, and disturbance of equilibrium. He was unable to walk and was brought in a wheel chair. He had had severe pain in the right ear for five weeks and severe temporal headache for about two weeks. His temperature was normal. He had a stiff neck. His spinal fluid showed an initial pressure of 200. There were 115 cells in the spinal fluid, 80 per cent of which were polymorphonuclears. Sugar was present. No organisms were seen on direct smear. He had a foul discharge coming from a marginal perforation. He required two doses of morphine sulfate to control the headache that afternoon. His cranial nerves were intact except for the eighth nerve, which showed complete loss of acoustic and vestibular function. The caloric test with cold water showed no response, and he was found to be stone deaf when his hearing was tested with the noise apparatus in the good ear. He did not show symptoms of fistula. He was too sick to be turned. He had spontaneous first degree nystagmus to the left side.

DISCUSSION

DR. A. S. MACMILLAN: Roentgenograms showed sclerosis of both mastoids, with no destruction. The lateral sinus is well forward. The region of the antrum is not enlarged.

DR. P. MYSEL: I started operating with the hand drill, but as the cortex was very thick and hard I decided to give up the hand drill and resorted to the mallet and gouge. The patient had a large perisinal abscess. Culture showed no growth. I worked on this man in my own way. I took off the whole cortex, which gave me a shallow crater to work in. I then started to work forward from the anterior edge of the sinus. I removed Trautmann's triangle. The patient had many granulations in the middle ear and in the region of the solid angle. I removed these. By working forward with the rongeur, I was able to enter the posterior external canal and the vestibule from behind. I took off the promontory. In making a tunnel under the external canal I could see that the canal cracked and started to be movable. The bone of the posterior canal was necrotic. I took off the whole external canal and exposed the facial nerve from its entrance to the middle ear down to the stylomastoid foramen. I did not open the sheath.

The next day the headache was gone. The patient has had an uneventful convalescence. But in spite of being able to close his eye much better, he shows a reaction of degeneration already.

DR. H. P. MOSHER: If there is already a reaction of degeneration, why not slit the sheath?

DR. P. MYSEL: I am waiting for the middle ear and the suppuration to clear up. I intend to do something of that sort in a week or two, when I am sure nothing more is going to turn up.

DR. H. G. TOBEY: I would rather wait. I think the nerve will come back.

DR. H. P. MOSHER: I like the idea of getting a wide operative field when one can. It is especially advantageous for procedures on the neck—getting a big enough incision so that one can get what one is working on as superficial as one can.

DR. P. MYSEL: It is much better in these operations to expose the sinus and work forward.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Regular Monthly Meeting, Dec. 6, 1937

WALTER H. THEOBALD, M.D., *President*

OTOLARYNGOLOGIC MANIFESTATIONS OF HEMOPHILIA. DR. CARROLL L. BIRCH (by invitation).

Hemophilia is usually classified under the hemorrhagic diatheses. It is characterized by habitual hemorrhage from various parts of the body, which may be spontaneous or may follow injury. The hemorrhage is excessive and out of all proportion to the extent of the injury. The hemorrhagic manifestations usually begin early in life. In my experience with 107 patients with hemophilia, 30 per cent had hemorrhages within the first three weeks of life, while 85 per cent had hemorrhages within the first three years.

This disease is hereditary and is linked with sex in its transmission. Only males have the disease, but it is transmitted by the unaffected female. All the daughters of a hemophiliac become transmitters of the defect. Theoretically, 50 per cent of their daughters become transmitters like themselves. Hemophilia is never transmitted through the normal male and is never passed directly from father to son. The sons of hemophiliacs are normal, and all their descendants are normal.

The only constant laboratory finding is prolongation of the coagulation time of the blood. In general, the longer the coagulation time the more severe the hemophilia.

Hemorrhage may occur any place in the body, subcutaneously, intramuscularly, nasally or from the gums, throat, stomach or intestine. A common site is the kidneys, while the most characteristic location is the joints. More rarely, hemorrhage may arise from the lungs, or it may occur in the spinal cord or brain, resulting in paralysis. All the 107 patients observed had subcutaneous hemorrhages, and all but one had intramuscular hemorrhages. Hemarthroses were found in 85 per cent. Of the 16 patients who had not suffered any hemorrhages in the joints, only 6 were beyond the age of 12 years. Permanent deformities of the joints were found in 68. Of the 39 who showed no crippling, only 9 were older than 12 years. When the disease is mild, permanent crippling is the result of repeated hemorrhages into the same joint and frequently occurs during the second or even the third decade of life. With these facts in mind, one realizes that the percentage of hemarthrosis and deformity appears to be lower than it actually is. The distribution of other hemorrhages was as follows: Forty-two patients had renal hemorrhages; 37 had peritoneal, gastric or intestinal hemorrhages; 101 had nasal hemorrhages; 98 had hemorrhages from the mouth, including those from the teeth, gums and tongue; 6 had hemorrhages into the brain or the spinal cord, and 5 had pulmonary or pleural hemorrhages.

Subcutaneous and intramuscular hemorrhages assume serious proportions in dangerous areas, such as the head and the neck. Many are traumatic, like those from biting the tongue or the inside of the cheek. Bleeding from the gums may be caused by the eruption or loss of the baby teeth, by foreign or hard particles in food, such as fish bones or nuts, or by the bristles from a hard tooth brush. In early childhood biting on hard sharp toys is a frequent cause of serious hemorrhage in the mouth. Falling with a pencil or other article in the mouth is not a rare cause of fulminating loss of blood. Infection, pyorrhea or acute tonsillitis is often accompanied by extensive hemorrhage into the floor of the mouth, into the tongue and down into the neck. Sometimes bleeding into the tissues of the throat and neck becomes so extensive that swallowing is impossible and breathing difficult. Other causes for hemorrhage into this area are abscessed or impacted teeth. The patient with a hemorrhage in the head presents a pathetic picture. The swelling and discoloration extend up into both eyes; the lids become so swollen that the eyes cannot be opened and the tongue so enlarged that the

mouth is held open and the saliva cannot be swallowed and drools from the corners of the mouth. Although it has seemed at times that the pressure must be relieved, I have always refrained from surgical intervention, and to date all of the patients have recovered. The lantern slides illustrate the severity and the extent of such hemorrhages in the head and the neck much more graphically than words.

Epistaxis is almost universal in patients with hemophilia. It may take the form of small, frequently recurring hemorrhages, or the patient may have severe, depleting loss of blood. In 7 of the patients observed epistaxis was the earliest recalled hemorrhage.

In 22 patients the initial hemorrhage occurred from the mouth (including the tongue, lips and gums). Thus, approximately 30 per cent of hemophiliacs have initial symptoms which would direct them first to the specialist in diseases of the ear, nose and throat or to the dentist. When it is remembered how serious surgical intervention is in patients with this disease, the responsibility of the first consultant becomes apparent.

In a survey of 113 deaths due to hemophilia it was learned that 57 per cent of the patients died before they reached the fifth year of life. Twenty-five per cent of the deaths were caused by surgical procedures; 15 per cent were the result of circumcision; 6 per cent followed extraction of teeth, although the diagnosis was known in each instance before the tooth was extracted; 1 death resulted from tonsillectomy, 1 from lancing the throat to relieve the pressure symptoms of hemorrhage and 1 from lancing hematoma of the scalp. One death followed vaccination; this is the only occurrence of hemorrhage after vaccination, even though all but 1 of the patients over 7 years of age have been vaccinated. Small accidental cuts were fatal in 23, and 6 had fatal epistaxis. There were 3 fatal hemorrhages from the throat during the course of diphtheria. One patient bled to death from the gums while cutting a baby tooth. Two patients died of natural causes, while the remainder died of hemorrhages elsewhere in the body.

From this brief survey it is plain that hemophiliacs are subject to frequent and severe hemorrhages in the region of the head and neck and that otolaryngologists have a definite responsibility to them.

DISCUSSION

Dr. Katsuji Kato (by invitation): This presentation of hemophilia by Dr. Birch is extremely interesting. I have never before seen such a complete group of pictures illustrating the various manifestations of this important disease. Since I am interested primarily in various types of diseases of the blood in infants and in children, I may speak of one or two matters which are uppermost in my mind.

Theoretically, pediatricians should encounter many cases of hemophilia; the bleeding tendencies of hemophiliacs are more apt to be manifest in childhood, because of a greater possibility for trauma in this period than in adult life. According to my experience, however, the incidence of the disease is low. This may be due to the fact that the diagnosis is missed, not so much because of a lack of interest in the disease but rather because of the lack of a simple method of estimating the coagulating time of blood that is easily applicable to infants and children. I suppose the most reliable method, either that of Lee and White or that of Howell, is to use venous blood, but venipuncture is not as readily practicable for children as for adults. To be sure, venous blood can be obtained by the puncture of the superior longitudinal sinus or of the external jugular vein in infants, but it is so obtained only in exceptional cases.

In routine work, however, capillary blood must be used. I have been for some time using a coagulation test on the capillary blood, in spite of the objections often raised. I use a hanging drop slide with a central depression, about 15 mm. in diameter and 3 mm. in depth. Having coated the surface of this depression with a small amount of melted paraffin, I take the slide to the bedside. From a clean deep puncture wound of either a finger or an ear lobe, I allow 2 or 3 drops of blood to fall into the paraffin-coated depression, in which a small clean glass bead (3 mm. in diameter) is immediately placed. After covering with another

slide and closing the depression cavity with petrolatum, I tilt the slide from time to time. When the glass bead becomes immovable as the slide is tilted in a vertical position, the coagulation is regarded as complete. Dr. Hayden stated that it is a waste of time to try to determine the coagulation time with capillary blood, but for the reason stated I use this method as a matter of routine. Whenever an abnormally prolonged coagulation time is obtained, a check must of course be made on the venous blood. For routine purposes, however, I find this capillary test to be fairly satisfactory.

Another matter of tremendous importance is the fact that the exact mechanism of the delayed coagulation in hemophilia is not as yet known. The problem was investigated recently by Bendien and van Creveld, of Holland (1937). They precipitated a certain fraction of globulin from the normal blood serum and noted that this fraction of the protein (called coagulation globulin), when administered intramuscularly or otherwise, will reduce the coagulation time of hemophilic blood. I am not in a position to discuss the chemistry of their investigation, but it seems to offer a great promise in the future studies of the mechanism of coagulation. Dr. Birch in 1933 aroused a great deal of interest in the treatment of hemophiliacs by the use of estrogenic substance, which has subsequently been tried by a number of observers, some with good and others with poor results. Whatever theory of coagulation one adheres to, one should not cease to search for the real factor which characterizes the defect in hemophilic blood. I think that hemophiliacs can never be permanently cured, but by the proper use of remedies one can more or less correct the abnormalities of hemophilic blood or at least keep the coagulation time of the patient to the minimal level.

DR. T. C. GALLOWAY: The number of cases studied was amazing, and I should like to know from how large a clinical group they were collected. Many patients who come to the physician's office as bleeders are found on careful study not to be hemophiliacs. They have purpura, jaundice or some other disease that causes a prolonged bleeding time, but not hemophilia. I have seen one patient who was undoubtedly a hemophiliac, and there was severe bleeding from a puncture of the antrum. This man, a doctor, knew he was a hemophiliac, and he had previously had a severe hemorrhage after a slight injury. Packs and various other measures failed, but the bleeding was controlled without difficulty by desiccating diathermy. I wonder whether Dr. Birch has tried this remedy.

DR. FRANCIS L. LEDERER: Dr. Birch's time was very limited for a complete presentation of this subject, but here is a person who can make a romance out of a blood dyscrasia. I am sorry she did not have time to tell us more concerning the ulcerating lesions about the pharynx which accompany other disturbances of the blood. The largest percentage of patients come from the department of diseases of the nose and throat. Numerous lesions about the pharynx are encountered, particularly in association with purpura and leukemia, which she might have discussed had time permitted. The point Dr. Kato made about coagulation and the lack of dependability of the ordinary methods employed is important. I have a feeling that the test is a useless procedure. Coagulation tests are done as a matter of routine to satisfy the laity, and they may or may not mean anything, first because of technic and secondly because there is a quiescent interval in hemophilia, during which time the reaction to the test is normal. Dr. Birch might tell whether a hemophiliac would at all times show a positive reaction.

Concerning the number of cases, Dr. Birch is fortunate or unfortunate in having a reputation in this field, and many patients are sent directly to her from all over the country. I believe that in Osler's entire experience he saw but 60 patients. Dr. Birch has gone beyond the clinical scope, for she has been abroad, where she interrogated members of the royal families and studied the family trees in regard to hemophilia. A discussion of her findings may be of considerable interest to those present.

One should refrain from all vigorous efforts to control a hemorrhage locally. In view of Dr. Birch's experience, it is just as well to try the "hands off" policy,

giving transfusions as a supportive measure. The fact that in Dr. Galloway's case the hemorrhage stopped after diathermy leads me to believe that other significant factors must have been present. Dr. Birch makes the point emphatic, and it is well taken, that the lesions should not be disturbed. One should expectantly wait for the clots to come away instead of trying packs or any of the local procedures usually employed in controlling hemorrhage.

Dr. Irving I. Muskat: It has come to my attention in my reading that some good effects have resulted from the local application of mothers' milk. I should also like to know whether Dr. Birch has had any experience with estrogenic substances in the treatment of hemophilia.

Dr. Frank J. Novak: Will Dr. Birch give us her experience with the use of estrogenic substances and of extremely large doses of viosterol in the treatment of hemophilia?

Referring to the question that has been suggested in the discussion, Is a hemophiliac a hemophiliac always or are there times when he is not a hemophiliac: The questions that have been raised regarding coagulation time are suggestive, and I wish to hear what Dr. Birch has to say about the blood clotting methods commonly used.

I must disagree with Dr. Birch about the fact that specialists in diseases of the nose and throat may not recognize hemophilia. I have heard them accused of a great many things, but I think that any one of them would know a hemophiliac if he had operated on him, and the number, so far as I know, is small.

Dr. Alfred Lewy: I might report a case of hemophilia that I was unfortunate enough to observe. The patient was a man, aged 21, with a history of bloody effusion into the knee joints. His brother had had the same condition, and his uncle on the maternal side had died of a hemorrhage due to hemophilia. When I saw this young man he had what was thought to be a peritonsillar abscess. His temperature was 102 F., and he had had pain and dysphagia due to the obstruction for two or three days. There was already infiltration under the tongue. I hospitalized him without making any incision, because I was familiar with his history. During the night breathing became so obstructed that a tracheotomy had to be done. It was performed with the patient in a semirecumbent position, through about 2 inches (5 cm.) of infiltrated tissue. Every bleeder that could be recognized was picked up and tied and the tube was inserted, but blood continued to ooze from the wound. The patient died within twenty-four hours, I do not know whether of sepsis or from the bleeding. The whole front of his body was more or less covered with blood during the night, but apparently he did not die of loss of blood alone.

I wish to ask whether Dr. Birch has had any experience with the use of moccasin snake venom and with the use of fer de lance, the local application of which has recently been recommended for the control of such bleeding.

Dr. G. H. Scott: Repeated transfusions were found to be of great value in one case of hemophilia. The patient was a recognized hemophiliac, aged 24, and the extraction of several teeth was deemed imperative. Studies of the blood were made, and several suitable donors were procured beforehand. After the extractions blood oozed steadily from the gums, as had been anticipated. It was found that a transfusion would check the oozing for a more or less definite period, the length of time being similar in each instance. Transfusions were then spaced so that each consecutive one was given before the clotting effect of the one preceding had worn off. The method proved successful in this instance.

Dr. Carroll L. Birch: I would say, concerning methods of determining the coagulation time, that in cases of hemophilia venous blood should be used instead of capillary blood, for the reason that the admixture of any tissue juice will shorten the coagulation time. I made a long series of determinations using capillary blood and venous blood. The differences were great, and there seemed to be

no definite correlation between the two methods. The clotting time of the venous blood was always longer. For small children I use a 27 gage needle and a small syringe and in most instances take the blood from the antecubital vein. I use Horoell's method of determining the coagulation time. I take 2 cc. of blood from patients under 7 years of age and 5 cc. from the older ones. Only blood obtained by clean venipuncture and without bubbles should be used. After the vein is entered the tourniquet should be removed to prevent stasis. It is a rather simple procedure, but every slip in technic tends to shorten the clotting time. The coagulation time of the same hemophiliac varies from time to time. Many hemophiliacs have cyclic variations. In most instances the coagulation time is longest in April and in November, and the most serious spontaneous hemorrhages have occurred in the spring and the fall. In cases of mild hemophilia the clotting time may be on one occasion normal and the next week considerably prolonged. In such cases, too, cycles are present. In doubtful cases several determinations should be made at least a week apart.

I have never been able to substantiate the diagnosis of hemophilia in any of the women referred with that diagnosis. Most of them had chronic purpura. My patients have come from all over the United States. Twenty-five were from Chicago, 36 from Illinois and the rest from outside the state. Many patients have been referred with the diagnosis of hemophilia who actually had purpura, scurvy or leukemia.

I have used snake venom, and in some cases it works very well. Used locally after the extraction of teeth, it has been of some help. In the average case transfusion will control hemorrhage for forty-eight hours but sometimes not so long. There is a boy in the hospital now who in an automobile accident sustained a fracture of the left femur, two lacerations of the scalp, a deep cut into the palm, a large hematoma of the chin, a small cut inside the mouth and a hematoma of the right knee. He has been in the hospital for thirty-six days and has received fifteen transfusions.

As for electrocoagulation, I have used it in open wounds. I have not had any help from intramuscular hemorrhages, but for hemorrhage due to a cut, by which blood is being lost outside the body, I have used the coagulating needle with some success.

I have obtained results with whole ovarian extract but not with theelin. Some physicians have obtained the same results with placental extract. English physicians claim results with extracts of white of egg. As Dr. Kato said, the factor is probably a split protein. Saliva, pineapple juice, trypsin and all other proteolytic enzymes will reduce the time to normal. A proteolytic enzyme probably produces this result, but just what it is no one really knows as yet.

I have used mothers' milk, and it probably is the same substance, an enzyme or a split protein. All of these things will clot the blood outside the body, but when they are applied to a bleeding point, the clot forms but the blood wells up from beneath and washes it away.

THE CLINICAL APPLICATION OF THE GALVANIC FALLING REACTION. EDWIN J. BLONDER.

Two hundred and thirty-eight patients were tested for their galvanic falling reaction by standing on a balance board while their mastoid was stimulated with a small amount of galvanic current. Normal persons required from 1 to 2 milliamperes, rarely as much as 5 milliamperes. Persons with a hyperactive labyrinth required 0.5 milliampere. Among the 118 pathologic patients were 63 with epilepsy, 21 with intracranial neoplasms, verified at operation by Dr. Loyal Davis, 15 with deafness and 3 with basilar fractures of the skull. The galvanic falling test corresponded to caloric stimulation except in 2 patients with intracranial neoplasms. One had a tentorial tumor and the other a temporal meningioma, and in each the eighth nerve was only partially involved. These patients were inactive to caloric stimulation but reacted to galvanic stimulation of 5 and 8 milliamperes.

I concluded that the falling reaction obtained with a patient standing on the balance board during galvanic stimulation is probably due to otolithic stimulation and presented experiments on decerebrated cats which substantiated this theory. The galvanic falling reaction was accurate, consistent and reliable in 237 patients. I suggest that a large series of examinations may localize isolated lesions in the vestibular postural pathway.

DISCUSSION

DR. LEWIS J. POLLOCK (by invitation): Since the apparent correlation between dissociated function of the semicircular canals and specific localization of lesions in the brain stem has not been confirmed, it is important to study other methods of examining the function of the vestibular nerve.

There is some controversy concerning the site of action of electrical stimulation in producing galvanic falling. Some investigators believe that the current stimulates the end organ, others that it stimulates the vestibular nerve and still others that it stimulates the brain stem. There is also some difference of opinion as to whether the otolithic organ alone is stimulated or whether the stimulus acts as does rotational and caloric stimulation of the semicircular canals. It may be of interest to point out some of the differences between the response in muscle and nerve and that in the vestibular apparatus. In muscle and nerve the response to electrical stimulation occurs only in the make or break of the current and not during the continued flow except, under certain conditions, when an initial tetanus of short duration is produced. The vestibular response, on the other hand, requires the continued passage of a direct current and persists as long as the current flows. In muscle and nerve responses occur not only to direct current but to alternating current if the frequency is not too great. There is no response to alternating current in the vestibular apparatus.

It is well known that an adequate stimulus of muscle and nerve not only must be of sufficient strength but must last for a certain minimal time. When one determines the shortest duration of a stimulus which is effective at twice the voltage necessary to produce an effect at infinite duration, one obtains the chronaxia of a tissue. The supposed chronaxia of the vestibular nerve is known to be as much as forty times that of spinal nerve or muscle. This suggests that, since the responses of the labyrinth are quickly effective to slight changes of position of the head, one is dealing not with chronaxia of a tissue but with a physical change, perhaps in the endolymph, comparable to a change produced by rotation and caloric change.

When a mixed nerve is stimulated, response occurs in both motor and sensory functions. Although galvanic nystagmus is noted, it is of an entirely different order from that obtained by turning and is a lesser part of the response. Finally, a falling is produced by repetitive stimuli at a frequency far greater than that which elicits response in a nerve or a muscle, although asynchronous stimulation of many fibers may explain this. It would seem that the response of falling to galvanic stimulation is the result of changes produced in the end organ or the endolymph or both. There is an indication for further work both on the clinical application of this method and on its physiology.

DR. SHERMAN L. SHAPIRO: This paper represents an attempt at exploring a method of vestibular examination which has long been known but has had little clinical application. The author is to be commended for this reason as well as for his refinement of technic in applying the test.

Certain thoughts occurred to me as I listened to the presentation. Dr. Blonder's clinical material represents a large variety of cases. I do not know that he claimed any special value for the falling reaction as compared with galvanic nystagmus except for the ease of application. This claim, however, is not true if Frenzel glasses are used, for they greatly facilitate the test for galvanic nystagmus. I have been able to obtain this reaction in normal persons with amounts of current as low as 0.5 milliampere.

Another point to be cleared up is the value of Dr. Blonder's test as compared with the caloric reactions. I was interested to hear him mention 2 cases in which

there was no reaction by the caloric method and in which nevertheless falling occurred toward the anode. Such cases indicate a possibility that something may be obtained in a diagnostic way from the galvanic examination that cannot be obtained by the caloric test.

As to the theoretic phase, first I wish to agree with Dr. Pollock that the make or break is not the important factor in producing a galvanic reaction. Any one, for instance, who performs the test for galvanic nystagmus will note that it takes a little while for the ocular movements to appear, even when they are observed under the brilliantly illuminated Frenzel glasses. I do not know about the otoliths being the site of stimulation. Everything from the internal ear to the vestibular nuclei has been cast in this role by various authors. I can only state that in my own experience I have produced a good galvanic nystagmus, using larger amounts of current than ordinary, in a patient whose eighth nerve had been sectioned intracranially and who had a dead ear by all usual criteria.

I did not hear Dr. Blonder state the level at which the cerebrospinal axis was severed in his cats. It is rather astonishing that no reaction was obtained, since the Utrecht school demonstrated years ago that the ocular reflexes can be obtained even when only the abducens nucleus is still below the level of the section. It is now well established that the reflexes of the eye, the body and the extremities are not dependent on the integrity of the cerebrum for their occurrence.

In summing up, the work of Dr. Blonder possesses great interest for students of neuro-otology and should form a point of departure for further research.

Dr. Ellison L. Ross: Dr. Blonder is to be congratulated on his careful and extensive work. It tends to place his method of treatment on a par with the caloric and rotation tests that are more commonly used. It is a question whether the utriculus or the semicircular canals or the entire labyrinth is stimulated by Dr. Blonder's method.

Dr. Edwin J. Blonder: Dr. Pollock in his discussion has brought out much interesting and important information regarding galvanic stimulation.

With regard to Dr. Shapiro's statement about galvanic nystagmus, I have no controversy with those persons who can interpret galvanic nystagmus with such accuracy that it can be of definite clinical value. The literature contains many articles in which the authors have said that galvanic nystagmus is inconsistent and inaccurate for purposes of diagnosis, and I have quoted a number of these in my bibliography. I hesitate to place the same reliance on galvanic nystagmus as I would on caloric nystagmus or galvanic falling. I have found galvanic falling as dependable as the results of caloric stimulation.

Galvanic nystagmus and galvanic falling do not appear to be results of stimulation of the same portion of the vestibular system. Magnus and de Kleyn first demonstrated that when an animal with decerebrate rigidity is placed on his back with his nose 45 degrees above the horizontal, the extensor tonus is increased. This increase they localized to the otolithic apparatus. Pollock and Davis confirmed this experiment with animals decerebrated by the anemic method. Dr. Loyal Davis and I produced an animal with decerebrate rigidity similar to those produced by Magnus and de Kleyn. We varied the tonus by galvanic stimulation of the auditory bulb, thereby altering the influence of the otolithic apparatus as described by Magnus and de Kleyn and others.

There is no question but that galvanic nystagmus can be produced by stimulation of the eighth nerve. The work of Wilson and Pike and Neumann was cited. We feel that small amounts of current stimulate the peripheral apparatus.

Regarding the question of the level of decerebration, our animals had not undergone surgical decerebrations, like those performed by Magnus and de Kleyn. Pollock and Davis developed an ingenous method of producing decerebration by anemia at levels they desired by ligating the basilar and carotid arteries, which they checked by the use of dyes. The level of decerebration was determined by varying the point of ligation of the basilar artery. We included the sixth nerve in our preparations.

Dr. Ross brought up the question of whether a portion of or all the labyrinth was stimulated by galvanic current. From our work it appears that the portion of the vestibular system concerned with posture is more responsive to galvanic stimulation when a balance board is utilized.

ACUTE BULBAR POLIOMYELITIS TWO WEEKS AFTER TONSILLECTOMY. ALICE K. HALL.

This case is reported in order to suggest the possibility of acute bulbar poliomyelitis in a differential diagnosis of paralysis of the soft palate and to call attention to the fact that a patient with this type of disease may first consult an otolaryngologist. It illustrates also the possible mildness of the systemic manifestations even during the acute paralytic phase of the disease.

On Oct. 30, 1937, Richard A., aged 8 years, was examined at the Northwestern University Medical School, his complaint being a speech defect of two days' duration associated with regurgitation of liquids into the nose. His tonsils had been removed two weeks before, and he had been attending school for the past week. The striking finding was almost complete paralysis of the soft palate, associated with stiffness of the neck and spine. A presumptive diagnosis of bulbar poliomyelitis was made, and the patient was transferred immediately to the Municipal Contagious Disease Hospital, where he remained for the usual twenty-one days. He returned home on November 18 and according to the latest report, received two days ago, was up and about the house, having no difficulty in swallowing and only a slight speech defect.

While I have no desire to commit the folly of drawing conclusions from a single case, the occurrence of this infection twelve days after a tonsillectomy leads to a consideration of the possibility of surgical poliomyelitis comparable to surgical scarlet fever as a clinical entity. There had not been any cases at Wesley Memorial Hospital, where the patient was operated on, or in the school which he attended. My associates and I tried to trace the source of infection, but it was impossible to do so, since there are probably many carriers of this disease.

COMPARATIVE TESTS ON AUDIOMETERS. AUSTIN A. HAYDEN.

As unnecessary minimum requirements increase the cost and thereby, to some extent, limit the adoption of any apparatus designed for wide clinical use, it is desirable to determine the value of such requirements. In audiometers pure wave form, which does add greatly to construction costs, has been considered necessary, especially by laboratory workers. To determine its real value in actual practice, 30 persons who were moderately hard of hearing were tested with four audiometers. Three of these X (Sonotone), Z (Western Electric 6-A) and Western Electric 2-A, probably meet the requirements of the Council on Physical Therapy, while Y (Marvel) has an admittedly imperfect wave form. Western Electric 2-A was arbitrarily taken as a standard for comparison because of its long established (but unfortunately far from sufficient) use and the literature it has created.

The four audiometers were placed in a small inside room of an otologist's office. The doors and windows in this and adjoining outside rooms were closed during the tests. The average noise level was then reduced from 50 to 20 decibels. A thick rubber hood around the air receiver, with a similar cup over the opposite ear, further lowered the noise level, but to exactly what extent was not determined.

Audiometer X is provided with dials for octave (from 128 to 8192) as well as sweep frequencies (from 2500 to 17000). Intensity is calibrated in steps of 5 decibels. It is provided with an automatic self-compensating pointer. Audiometer Y has an octave, octave letter and sweep frequency range from 64 to 8192, with attenuation from 0 to 100 decibels at all frequencies. Audiometer Z has two dials, one for sweep frequency marked with eight indicated points from 128 to 9847. For bone conduction tests, a ring is placed over the intensity dial for recalibration.

When being tested, the patient responded by the usual finger or electric signal. The average of a minimum of three readings based on air conduction at each of the octave frequencies was taken to be the actual loss of hearing and charted as such on the individual audiograms and recorded. These three readings almost never varied from each other by more than plus or minus 5 decibels. The tests were made by three experienced observers. Rest periods were used as needed to minimize confusion due to fatigue of both the patient and the observer.

Fourteen of the 30 patients were found to have losses of hearing greater than the Western Electric 2-A audiometer could measure. For that reason their audiograms were discarded. The losses of the remaining 16, as shown by each of the four audiometers, were averaged and plotted. The average difference for all frequencies is readily compiled and is not great.

Summary.—Four audiometers of different makes were investigated to determine their comparative performances for air conduction tests and the need for pure wave form in clinical practice. Audiometer X consistently showed the highest losses of hearing; Y showed a medium but more erratic loss, and Z showed the smallest loss. As compared with the 2-A audiometer the average differences were plus 5 decibels for X, minus 3 decibels for Y and minus 6 decibels for Z. Audiometers X, Y and Z were capable of measuring greater losses of hearing than 2-A. Plus or minus 5 decibels was found to be the combined human error factor of observer and patient. An average quiet, not soundproof, room was used for the tests.

Conclusions.—Audiometers, despite the foregoing variations, furnish the best means of testing hearing acuity. Any one of the four tested will be more useful in clinical practice than any other means now available for testing and recording hearing acuity. The lack of purity of sound wave apparently did not introduce any serious errors that were not largely explainable by other causes. A quiet room is essential. The need for a sound-proof room increases as the loss of hearing to be tested decreases. An audiometer should be part of the office equipment of every otologist.

Otologists' Committee.—Through the Otologists' Committee on Audiometers and Hearing Aids,[1] the Council on Physical Therapy of the American Medical Association places its facilities at the disposal of otologists for the purpose of correlating clinical experience, laboratory procedure and manufacturing detail. Acceptance by this Council should become an incentive to manufacturers to construct instruments in accordance with agreed-on clinical requirements and should assure otologists that advertised claims meet required specifications.

DISCUSSION

MR. HOWARD A. CARTER (Secretary, Council on Physical Therapy, American Medical Association): As far as I am aware, this report is the first and only one in the literature on four audiometers tested concurrently. I have been asked to discuss it because I have had experience in this field with the Council on Physical Therapy, which has been aided by the committee mentioned by Dr. Hayden. In *The Journal of the American Medical Association,* Nov. 27, 1937, a report "Tentative Minimum Requirements for Acceptable Audiometers" was published. I have a reprint of that report in my hand, and physicians may obtain one by writing to the American Medical Association. I should like to review some of the points considered when these requirements were formulated. These requirements are the summation of the experiences of qualified men who have been working in this field for some time. Group opinion at times is difficult to deal

1. This committee is composed of Drs. George M. Coates, Lee Wallace Dean, Edmund Prince Fowler, Austin A. Hayden (chairman), Isaac H. Jones, Douglas MacFarlan, Burt R. Shurly, Horace Newhart and William P. Wherry. Mr. Howard A. Carter is secretary. Several of the individual members have availed themselves of the services of such physicists as Bunch, Fletcher, Hartig, Knudson and Rogers.

with, but when conclusions are reached they are usually reliable. Even for the definition of a clinical audiometer, difficulty was encountered. At the outset the consultants had a long paragraph for the definition, but when they had finished only one short sentence was left: "A clinical audiometer is an instrument for measuring the acuity and range of hearing."

It was felt by the consultants that audiometers should produce frequencies from 128 to 8192 cycles per second in sweep or in seven discrete frequency stops. The limits of the tolerable frequency variation are important, and familiarity with them may be most helpful to the otologist when salesmen come around with sales talks for audiometers. The frequency variation should not be more than plus 5 per cent at all frequencies. Audiometers should be calibrated in decibels and should not have more than 5 decibels to a step. The range limits for intensity, according to the requirements, should be plus 1½ decibels per 5 decibel steps. The term "percentage hearing" was ruled an inadequate method of expressing loss of hearing.

Much discussion was devoted to the wave form. It was the opinion of some of the consultants that the output should be a pure sine wave. Others said that an absolute sine wave is not necessary. If the wave form did not follow the sine wave, some believed that harmonics were present. It was felt that harmonics of a low tone might be interpreted as a high frequency note to a person deaf to low tones but only moderately deaf to high tones. Dr. Hayden has apparently shown that wave form does not have as much to do with the successful operation of the instrument as might be expected.

The auditory chart, or audiogram, was considered by the consultants, but no agreement was reached and no uniform chart recommended for adoption. It was felt that physicians may have adopted their own method of recording and might object to a uniform method of correlating data.

The definition of threshold of hearing adopted reads: "The threshold of hearing is the audiometer setting corresponding to the lowest intensity at which the person being tested is able to indicate correctly more than half the time that he is hearing."

The consultants recommended that the rules of the Council on Physical Therapy might well be adhered to by the manufacturers of acceptable audiometers so far as the marketing methods and advertising statements are concerned.

DR. T. C. GALLOWAY: What are the effects on audiometers of variations in voltage of the city circuits? Also, how may one know when a machine is out of order without testing it against some other machine? What are the other chances that an audiometer will get out of order without the fact's being detected?

MR. HOWARD A. CARTER: Some audiometers are provided with voltage regulators which help maintain a constant intensity as predetermined by calibration.

Audiometers are on the market equipped with a sweep frequency and discrete frequency steps. In case of audiometers equipped with a heterodyne circuit, in which one frequency beats against another frequency, unless provisions are made the resultant output frequency of the unit will vary from time to time. Hence, to make sure that the unit is calibrating correctly, an indicating light is fixed on the panel of some of the units; this turns on when the dial is correctly set at a predetermined known frequency.

If the audiometer is out of order it is highly probable that an experienced operator would know it. However, in some instances there may be only a slight disturbance; for example, inaccuracy of calibration with slight irregularities can be located only by examination and special tests.

DR. ALFRED LEWY: In the absence of Dr. Sonnenschein, I presume it devolves on me to say something about audiometers and tuning forks. It is refreshing to hear Dr. Hayden admit that audiometers vary. If he tried out several audiometers made by the Western Electric Company he would probably find that they also varied from each other. I agree that the audiometer is an adequate instrument for the measurement of the acuity of hearing. In addition it is of value in

medicolegal cases, particularly in catching malingerers who feign a partial loss of hearing. It is practically impossible for such persons to give the same graph on repeated tests.

At the last meeting of the American Laryngological, Otological and Rhinological Society, Inc., Dr. Ersner and a Mr. Podolsky, an acoustic engineer, presented a long and carefully prepared article on the results of their investigations of five of the leading makes of audiometer. Not one of them was accurate in all particulars. Every one had a fault which would disqualify it as an accurate scientific instrument. Moreover, while the audiometer measures the acuity of hearing, it is impossible to make a diagnosis in many instances without a determination of the bone conduction. Otherwise the manufacturers would not have gone to the labor and expense of developing an attachment and chart for recording the bone conduction. They now admit this but did not at first.

In regard to tuning forks, when one is measuring something that varies in caliber from time to time, as human hearing is known to do, one does not use a micrometer any more than an artisan does in measuring a piece of wood which is subject to change from moisture and temperature. I therefore agree that for practical purposes it is not necessary to have the audiometer so accurately calibrated. Neither should it be offered to members of the medical profession as an instrument of scientific precision. I should also like to know why the prices of audiometers vary so greatly when for all purposes demanded by the standards set by the American Medical Association each instrument will give the same data. Why does one cost $200 and another $600?

I wish to call attention to the fact that Professor Struycken, the inventor of the monochord, has brought out a tuning fork holder with a hammer activated by a spring which can be regulated to a standard intensity, so that the applied force can always be the same.

Tuning forks have been used for diagnostic purposes by several generations of otologists. Their uses and their faults are familiar. I still believe that I can make a good diagnosis with tuning forks so far as pathologic changes and indications for treatment are concerned.

DR. AUSTIN A. HAYDEN: Regarding Dr. Galloway's question of the variation of 2048, I had an audiogram made in St. Louis on my own ears, and on the instrument I think Dr. Galloway was talking about it showed a dip of only 10 decibels. Just why that is I do not know. It goes without saying that there is much about audiometers that is not known as yet.

Regarding Dr. Lewy's question about the different makes of the same instrument giving different readings, it depends on what is meant by "different readings." I think that most of them give rather constant trends in readings, but they do not give the same readings. I do not think one could expect them to, for there are so many variations in the air and in the noise level of rooms and many tests are made in rooms that are not sound proof. All of these factors cause inescapable errors. As I read Dr. Ersner's article I was struck by the fact that Dr. Ersner and Dr. Lewy might have talked the matter over together. The trends in the audiometers are similar; they are not exact and I think are not expected to be exact.

About diagnosing pathologic lesions without testing the bone conduction, no audiometer is put out now without an attachment for this purpose. The addition was not made, I suspect, on account of the diagnosing of diseases of the ear, but rather to facilitate the fitting of hearing aids when a bone conduction apparatus will work better than an air conduction apparatus.

About the cost of audiometers, Dr. Lewy wants to know why they are so expensive. I wanted to know that myself. I paid $640 for the 2-A Western Electric audiometer I bought several years ago and am told that on every machine the company sold it lost money. There is a tremendous resistance to these instruments; possibly more resistance in Chicago than elsewhere. Just as any apparatus will be reduced in price with more general use, so will the audiometer. The manufacturers say that the variations depend on the construction of the machine.

I thoroughly believe that hearing acuity will be measured by audiometers in the future and that persons who do not have audiometers are not in a position to prescribe hearing aids and to practice otology. The answer to the difference in the cost of various machines is the difference that manufacturers figure in regard to their machines. There has already been a considerable reduction in the price. I believe that at present the range in price is from $200 to $300, and I think that any of the machines that I have discussed tonight will do satisfactory work. I think that there are some others which will do good work, but I have not had a chance to investigate them.

Book Reviews

Surgical Anatomy of the Head and Neck. By JOHN FINCH BARNHILL, M.D., F.A.C.S., LL.D., with an introduction by Paul S. McKibben. Price, $20. Pp. 931, with 431 illustrations. Baltimore: William Wood & Company, 1937.

The author of this splendid work is one of the outstanding otolaryngologists of the United States. His service in this specialty from the time he first began his career has been of such high quality that he quickly received the proper recognition. Eminently successful as a clinician and a surgeon, he accepted important teaching appointments, which as time went on endeared him to an ever increasing number of pupils, both here and abroad. In addition, he contributed much to otolaryngologic literature, having written several valuable textbooks and numerous articles. His papers and discussions presented before the national laryngologic societies will linger long in the memories of those whose privilege it has been to listen to him.

In the course of time Dr. Barnhill has received many honors. A glance at the list on the title page of his book showing his connections with university faculties as well as the leading national laryngologic bodies of which he is a member convinces one that he has well merited those honors.

It is well known that early in his career he recognized the fundamental importance of anatomy and took pains to perfect his knowledge of the regions in which he worked, as Professor McKibben states in the introduction of this book. In the late 1890's, while attending a seminar in a postgraduate hospital in London, Dr. Barnhill was stunned by the remark of a well known English laryngologist when a certain anatomic question came up for discussion and the Americans present showed pitiful ignorance of it. The English laryngologist remarked: "Our American students know less anatomy than those from South Africa."

Dr. Barnhill felt that this remark, though perhaps justified, was due principally to a state of mind existing in England at that time.

The English and American governments had been in a violent dispute over Venezuela, and President Cleveland had just sent an aggravating, defiant message that had stirred Great Britain to her political depths, and to humiliate America was just then thought patriotic. This remark, however, clung to Dr. Barnhill, and stung him, and constituted the germ from which this volume grew.

The author maintained a heavy schedule, which consisted of teaching medical students the principles of otolaryngology, giving courses in surgery of the head and neck, taking an active part in the leading national laryngologic bodies, contributing to the literature of this specialty and carrying on consultation and office practice. His course on the surgical anatomy of the head and neck attracted students from all parts of this country. It became more and more systematized, and Dr. Barnhill was requested to offer, every now and again, courses in different centers. Indeed, his course was eagerly sought after by numerous students.

Though he had the sum and substance of his well organized course systematically arranged in notes and had for some time considered putting the material in book form, this idea did not materialize until his many students persuaded him finally to put it in a volume.

This work therefore represents Dr. Barnhill's postgraduate course in the surgical anatomy of the head and neck.

The text is so arranged that detailed instruction is given in each and every region of the head and neck. Each period is devoted to a special region: Period 1 takes up the neck and period 2 continues with the neck. Period 3 details the carotid arteries and the internal jugular veins, and period 4, the recurrent laryngeal nerves and the inferior thyroid artery, while period 5 takes up the thyroid and parathyroid glands. The author showed great anxiety that the student should clearly identify the important structures contained in the head and neck; he divided his periods in such wise that the important points of anatomy are thoroughly worked out.

The surgical anatomy of the middle ear is given in one period, that of the mastoid in another and that of the petrous pyramid in another. The various air sinuses are detailed, one period each being allotted to the maxillary sinus, the sphenoid sinus, the ethmoid cells and the frontal sinus. The various structures in the orbit are adequately covered in two different periods. The brain, the meninges, the important nerves and ganglions, as well as all the fossae, are fully discussed; the nose, the pharynx, the mouth, the tongue and the teeth, the floor of the mouth, the larynx and the trachea and surgical treatment of these structures are fully dealt with in the properly assigned periods, which the author has organized, it seems to me, with fine integration.

Period 33 is entirely devoted to the subject of local anesthesia. The instructions show painstakingness, for the author has left nothing undone to explain every minute detail. The advantages and disadvantages, apprehensive patients, the necessary instruments, methods (infiltration and block), as well as the complete technic of anesthetizing the various structures in the head and neck, all important points, are clearly set forth. This period alone is one of the most practical guides available to efficient production of local anesthesia. It is needless to emphasize how important a knowledge of local anesthesia is to otolaryngologists, oral surgeons, dentists and general surgeons. This chapter will ever be a useful one to them.

The first half of this volume is therefore a careful review of all the regions of the head and neck. It guides the student in making dissections and points out the significance and the importance of the structures. Such practical points as infection and its extension, as well as surgical intervention on the structure under consideration, are brought out again and again. This review is without doubt one of the finest contributions which has been made.

The second half of this volume is taken up with illustrations, totaling 431. Many of these are in fine color, and are replicas of the preparations that the author or his students have made. The illustrations are arranged parallel with the didactic text. They augment the value of the text by bringing before the student the picture corresponding to the word. To bring out so much detail, it was necessary to use a great number of specimens. Often when different parts of a structure needed special study, more specimens were dissected for that structure; this was done, for example, with the petrous pyramid, the origin, course and distribution of the cranial nerves, the nasal accessory sinuses, the larynx and intricate nerve pathways in the nose, throat and ears.

Here and there the author added certain schematic drawings depicting the reason for certain reflexes, for example, cough due to irritation of the external ear and the course of reflex pain from the throat or decayed tooth to the ear. Numerous other schematic drawings show vividly the anatomic features and distribution of important ganglions and the line of incision used in the standard operations on the ear, nose, throat and other structures of the head. Still other diagrams are seen which add further to the value of the entire text, for these depict fine points in connection with the surgical procedures for the various structures of the head and neck, aids which are often hard to find in other standard works of anatomy and surgery.

Many of the illustrations are so valuable that they indeed represent a complete study in themselves; for the seeker after the truth is certain to spend a great deal of time reviewing them, to the end that they will become a "nerve cell habit" with him. Special problems of anatomy and surgery often come up with the numerous conditions encountered in the head and neck, and it may well be that a solution to many of these can be found by a careful study of the special period and the corresponding illustrations dealing with that problem.

As one reviews this book several times, newer concepts and ideas permeate one's mind. This volume is as close an approach to living anatomy and living surgery of the head and neck as one can find in book form.

It is earnestly recommended to otolaryngologists, ophthalmologists, general surgeons, neurosurgeons, neurologists, oral surgeons, graduate students in the various specialties of the head and neck, dentists and students of medicine and of dentistry.

Directory of Otolaryngologic Societies *

FOREIGN

COLLEGIUM OTO-RHINO-LARYNGOLOGICUM AMICITIÆ SACRUM

President: Prof. H. Burger, Keizersgracht 317 C, Amsterdam.
Secretary: Prof. Dr. C. E. Benjamins, Verlengde Heereweg 143, Groningen, Netherlands.

SOCIEDAD RIOPLATENSE DE OTO-RINO-LARINGOLOGÍA (ARGENTINE SECTION)

President: Dr. Raul Becco, B. Mitre 1690, Buenos Aires, Argentina.
Secretary: Dr. Juan Manuel Tato, Santa Fé 1171, Buenos Aires, Argentina.

NATIONAL

AMERICAN MEDICAL ASSOCIATION, SCIENTIFIC ASSEMBLY, SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY

Chairman: Dr. Gordon B. New, Mayo Clinic, Rochester, Minn.
Secretary: Dr. Leroy A. Schall, 270 Commonwealth Ave., Boston.
Place: San Francisco. Time: June 13-17, 1938.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Harry S. Gradle, 58 E. Washington St., Chicago.
Executive Secretary: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.
Place: Washington, D. C. Time: Oct. 9-14, 1938.

AMERICAN BRONCHOSCOPIC SOCIETY

President: Dr. Gordon Berry, 36 Pleasant St., Worcester, Mass.
Secretary: Dr. Lyman Richards, 319 Longwood Ave., Boston.
Place: Atlantic City, N. J. Time: April 30, 1938.

AMERICAN LARYNGOLOGICAL ASSOCIATION

President: Dr. J. F. Barnhill, Miami Beach, Fla.
Secretary: Dr. James A. Babbitt, 1912 Spruce St., Philadelphia.
Place: Atlantic City, N. J. Time: May 2-4, 1938.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

President: Dr. Samuel J. Kopetzky, 71 E. 80th St., New York.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester, N. Y.
Place: Atlantic City, N. J. Time: April 27-29, 1938.

SECTIONS:

Eastern.—Chairman: Dr. Louis H. Clerf, 1530 Locust St., Philadelphia.
Place: Philadelphia. Time: Jan. 7, 1938.
Southern.—Chairman: Dr. Murdock S. Equen, Medical Arts Bldg., Atlanta, Ga.
Place: Atlanta, Ga. Time: Jan. 24, 1938.
Middle.—Chairman: Dr. James B. Costen, Beaumont Bldg., St. Louis.
Place: St. Louis, Mo. Time: Jan. 26, 1938.
Western.—Chairman: Dr. Arthur C. Jones, 105 N. 8th St., Boise, Idaho.
Place: Santa Barbara, Calif. Time: Jan. 30-31, 1938.

AMERICAN OTOLOGICAL SOCIETY

President: Dr. Harris P. Mosher, 828 Beacon St., Boston.
Secretary: Dr. Thomas J. Harris, 104 E. 40th St., New York.
Place: Atlantic City, N. J. Time: May 5-6, 1938.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.